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BUREAU OF HYGIENE AND TROPICAL DISEASES.

TROPICAL DISEASES BULLETIN.

Vol. 37.] 1940. [No. 12.

PLAGUE.

Précis of Abstracts in this Section.

Hampton (p. 822) traces the history of plague in the United States and details the large number of wild rodents which have now been found to be infected. Miller (p. 823) thinks that wild rodents of western Canada must eventually become infected with plague owing to the extension of sylvatic plague in the United States, and shows that a gopher, *Citellus r. richardsonii*, has been found infected in Alberta.

Sáenz Vera (p. 824) shows that in parts of Ecuador plague occurs in recurrent epidemics which are usually preceded by epizootics in rats and guineapigs. Field rodents constitute the reservoir and the link between them and man is the cui. The disease is essentially rural and there have been no alarming epidemics, owing probably to the fact that the rats are found in fields of sugar cane where food is abundant. Pinotti *et al.* (p. 824) record a small epidemic in a village near Rio de Janeiro, which was preceded by an epizootic in rats. They consider, however, that this was not an exacerbation of an enzootic but that the infection had been introduced from Rio de Janeiro.

Lamborn (p. 824) in Nyasaland has found that in an area where there occurred an outbreak of plague, a mild enzootic among field rodents had been in existence for some time.

Sokhey (p. 825) shows that *P. pestis* has a definite capsule, but he does not recognize what has been regarded by other workers as the "envelope." The capsule is present in both virulent and avirulent strains, and the antigenic value of the organism does not seem to make any difference in that the capsule is present in strains of both high and low immunogenic power.

Rao (p. 825) has investigated the capacity of *P. pestis* to oxidize various substances. Carbohydrates, especially hexoses, certain organic acids and amino-acids are oxidized and it has been found that, for culture, glucose and lactic acid are the best sources of carbon. A list of the useful amino-acids is given. The same author (p. 826) has studied the effect of certain growth factors on the metabolism and growth of *P. pestis* in culture in media of known chemical composition. Four factors, haematin, cozymase, thiamin and nicotinic acid, stimulate respiration and reduce lag in growth. It may be that these substances are essential components of the bacterial cell, synthesized during growth, but that if they are made readily available, rapid growth is facilitated. Wats and Puduval (p. 827) have obtained smooth and

rough strains of *P. pestis* and have investigated the soluble substance obtained in washings of growth. The presence or absence of this substance has no relation to virulence but its titre seems to indicate the antigenic value of a strain.

In the examination of adult *Mastomys ugandae*, the domestic rat of the Lake Albert region, DEVIGNAT (p. 828) aspirates the bone marrow of the femur, suspends it in saline and inoculates young rats. From the results of this investigation, the endemic area has been defined. It is pointed out that apparently healthy rats may show infection.

LEWILLON *et al.* (p. 828) record a case of meningitis due to *P. pestis*.

SOKHEY and DIKSHIT (p. 828) have found that sulphathiazole is much better than sulphapyridine in the treatment of experimental plague in mice, and effected cure in 80 to 90 per cent. of animals treated. They propose to try this drug in human cases. MOREAU (p. 829) reports recovery of two cases of bubonic plague treated with subcutaneous injections of E.V. serum and the sulphonamide preparation "bacteramide" by mouth.

GIRARD (p. 829) refers to the recurrences of plague which may take place after long periods of freedom from the disease. Epizootics in rats occur in the areas of Madagascar referred to and since destruction of fleas in houses with earth floors and thatch roofs is not possible, the author considers that efficient vaccination of the population is the best measure of prevention against plague. GIRARD and RADAODY-RALAROSY (p. 830) show that bacilli of the avirulent E.V. strain may be found in the spleen and liver of guineapigs which have received large injections, but for a limited period only.

In the Annual Report of the Board of Health of Hawaii (p. 830) is a description of the preparation of banana-phosphorus bait for rats. AHLUWALIA (p. 830) states that cyanide preparations are better than trapping or baiting in anti-rat campaigns because they also destroy fleas, and they are superior to CO or SO_2. Calcid briquettes are more efficient and economical than Cyanogas but need to be used with proper control on account of their dangerous nature. PARANJOTHY (p. 831) found that arsenious oxide and barium carbonate were the cheapest of a number of poison baits tried. He (p. 831) states that herd experiments with strains of Salmonella and Pasteurella have failed to precipitate an epidemic in rats; Ectromelia is similarly unsuccessful.

JUNIOR (p. 832) describes the services carried out at the plague laboratory in São Paulo. *C. W.*

HAMPTON (Brock C.). **Plague in the United States.**—*Public Health Rep.* 1940. June 28. Vol. 55. No. 26. pp. 1143–1158. With 1 fig. [13 refs.]

In almost all countries the date of the first introduction of plague is rather ill defined. This seems due to the fact that rat plague has been the first type of infection to occur and that "rat plague may exist in a city for some time without the development of the disease in human beings." Another cause of the inability to give exact dates for the United States was the denial of the existence of plague for months "by many intelligent and well meaning but uninformed persons—some of them doctors and health officials—as well as by others whose action was probably based on commercial interests."

The first record of the appearance of plague in the United States falls to San Francisco, California, on March 6th, 1900, with the notification of death of a Chinaman. It was proved plague bacteriologically and by animal inoculation. The world pandemic of plague had begun in the Yunnan province in China 6 years previously. In San Francisco there occurred 121 cases with 113 deaths and the last case of this outbreak was recorded in February 1904. This last case, however, was not the end of the infection, although the epidemic had undoubtedly been brought to an end by vigorous cleansing and anti-rat operations, human plague was again discovered in San Francisco in May 1907. "During the period 1908–1914 plague appeared in other localities in California with a total of 22 human cases." The history of plague since its introduction on the east coast of the United States has been one of extension and up to January 1st, 1940, "there have been recorded 499 cases with 314 deaths." The human cases have been reported in eight States. It might be thought that as no more than 8 cases of human plague have been reported from 1930–1939 this disease had become one of trivial importance. This view, however, would ignore the danger of spread of sylvatic plague. Within 10 years after plague first appeared in San Francisco the infection was proved to be widespread among ground squirrels in nine Californian counties and all the evidence now available goes to show that plague has spread from rats "first to the ground squirrels and then to other wild rodents in western United States." It has also been shown that "wild rodent plague may spread unnoticed over great areas . . . and may continue to spread unless suppressive measures are adopted to prevent it." This sylvatic plague may be accompanied by little human plague, but with its approach to the large rat populations of industrial cities the danger of explosive and epidemic character is greatly increased. "To January 1, 1940, plague infection has been demonstrated in 14 species of ground squirrels, in red squirrels, tree squirrels, and flying squirrels, in wood rats, kangaroo rats, field mice, prairie dogs, chipmunks, marmots and a cottontail rabbit in western United States, and in fleas, lice, and ticks from wild rodents." This article ends on the hopeful note: "With full knowledge of how to prevent and control the disease, however, plague in epidemic form should never again be permitted to occur in any locality in the United States."

W. F. Harvey.

MILLER (Max J.). **Plague: History and Epidemiology.**—*Canadian Jl. Comp. Med.* Gardenvale, Quebec. 1940. July. Vol. 4. No. 7. pp. 183–193. With 1 map.

The main part of this article gives an excellent account of the history of plague from earliest times. It also deals with the discovery of the plague bacillus, the extensions and extent of present-day plague, and the epidemiology of the disease. So far as concerns Canada the author points out that "with gradual extension of sylvatic plague areas in the United States it could only be a matter of time when the wild rodents in western Canada acquired plague infection." Already a "gopher," *Citellus r. richardsonii*, has been found suffering from plague in Alberta. Unless immediate measures are taken it seems probable that sylvatic plague will spread widely through western Canada.

W. F. H.

SÁENZ VERA (C.). La peste en la provincia de Loja, Ecuador. [**Plague in the Province of Loja, Ecuador.**]—*Bol. Oficina Sanitaria Panamericana.* 1940. July. Vol. 19. No. 7. pp. 661–667.

Loja, in the southern part of Ecuador where it abuts on Peru, was surveyed in 1939 and found to have been the seat of recurrent epidemics of plague, usually preceded by epizootics among rats and guineapigs. It seems probable that the reservoir of plague has been the field rodent and that the link between those rodents and man has been the cui [a small rodent of the hamster type. The name is used for *Microcavia australis* and for *Graomys griseoflavus*]. Plague infection has been known in the province for 20 years, is endemic, and is essentially rural. This rural character and comparative absence of any alarming epidemic character is ascribed to the fact that the rat population is found in fields of sugar cane and yuca where food is abundant ; there is no necessity for the field rodents to resort to inhabited centres in search of food. Any attempt at eradicating plague from the province of Loja would be extremely difficult and costly. It would be next to impossible to exterminate the cuis ; the people are little subject to sanitary control and they live in close community with animals, especially pigs and cuis. Roads and means of transport are bad. Some suggestions are made for remedying the state of affairs, which naturally involve the expenditure of government money.

W. F. H.

PINOTTI (Mario), GENOFRE (Werneck), ROMEU (L. Barbosa) & VIANA (Miguelote). Casos de peste em Miguel Pereira. [**Plague in Miguel Pereira.**]—*Arquivos de Higiene.* Rio de Janeiro. 1939. Feb. Vol. 9. No. 1. pp. 117–165. With 2 folding plans. English summary (9 lines).

The village of Miguel Pereira is in the State of Rio de Janeiro and was attacked by a small epidemic in a population of 797 inhabitants. Almost every detail concerning the sector affected is described, down to the number of particular shops—barbers, carpenters, chemists, etc.—and the whole lay-out of the place. Altogether, 11 cases of plague occurred with 6 deaths and this epidemic was preceded by a rat epizootic. This epizootic, which centred in and around a grocery warehouse, was the cause of the first human cases among the employees. In the analysis of all the circumstances the authors conclude that the epizootic was not merely an exacerbation of an enzootic but had been introduced from Rio de Janeiro. Numerous tables and explanatory maps are given and, as is customary with papers in this journal, a summary in English is added on perforated board ready for filing as an abstract.

W. F. H.

OUTES (Julio D.). Consideraciones sobre el problema de la peste en el interior del pais. [**Plague in the Interior of the Argentine.**]—*Bol. Sanitario.* Buenos Aires. 1939. Oct. Vol. 3. No. 10. pp. 636–639.

LAMBORN (W. A.). **Investigations in Connection with an Outbreak of Plague.**—*Nyasaland Protectorate Ann. Med. & San. Rep. for Year ending 31st December, 1939.* Section VIII. pp. 27–28.

The investigation was carried out in the neighbourhood of a village in a subdistrict of Blantyre, where three human cases had occurred.

Confirmation of the clinical diagnosis was obtained by post mortem examination of a domestic rat. Dethatching of huts and grain stores permitted examination of 275 rodents of the species *Rattus rattus alexandrinus*, *Mastomys coucha* and one field mouse *Saccostomus campestris*. Some of these were living and some dead. The operations were extended also to fourteen villages within a radius of five miles. An attempt was made to trace the origin of the outbreak and it was discovered that a mild enzootic among field rodents had evidently been in existence for some time over a wide area. Ectoparasites were collected and arrangements were made to enlist the co-operation of headmen and villagers in a survey of the extent of the disease among wild rodents. *W. F. H.*

SOKHEY (S. S.). **The Capsule of the Plague Bacillus.**—*Jl. Path. & Bact.* 1940. July. Vol. 51. No. 1. pp. 97–103. With 22 figs. on 3 plates. [17 refs.]

The capsule of a bacillus has attained considerable importance, apart from its morphological diagnostic characters, because it is regarded as having antigenic property. In the case of the plague bacillus there seems to have been some tendency to distinguish both a capsule and an "envelope" and to assign antigenic value to the latter. The author in this paper "is solely concerned with an objective study of the capsule," but probably the most interesting part of the communication, which has to be read in the light of the controversy over the use of broth or agar plague vaccines and incubation at 37°C. or 27°C., relates to the antigenic values of the plague bacillus in different circumstances. It may be said in the first place that staining with Pitfield's flagellar stain and with Churchman and Emelianoff's stain show the plague bacillus to be definitely a capsulated organism. An organismal capsule is defined as: "A structure which surrounds the bacterial body, is not stained by the ordinary bacteriological dyes but only as a rule by special methods involving the use of mordants, sometimes appears as a clear unstained area standing out against a stained background (negative staining), seems to possess a confining membrane and often enlarges on contact with the animal body." Some of the findings of the author bearing on antigenic value of the plague bacillus are :—

1. "The capsule is present whether the organism is virulent or avirulent. The antigenic value of the organism does not seem to make any difference." 2. "The size of the organism is considerably affected by the temperature and duration of incubation and the medium. The organism as a whole, including both soma and capsule, is much larger when grown at 37°C. than at 27°C. or lower." 3. "The 'envelope' . . . appears to be nothing but . . . a peculiar settling down of the fine particles of India ink at a distance from the capsule due to the operation of physical forces."

W. F. H.

RAO (M. Sadashiva). **Oxidations effected by the Plague Bacillus.**—*Indian Jl. Med. Res.* 1940. Jan. Vol. 27. No. 3. pp. 617–626. With 3 graphs.

The purpose of this research has been to determine the energy sources available for the plague bacillus in nutrient media and the test of that availability has been the rate of oxidation of the substrate by a suspension of the dried organism in normal salt solution. Oxidative

activity was measured manometrically in air at 27°C. and 0·05 M. substrate concentration. Altogether 48 compounds were tested as substrates. "Oxidation is indicated and measured by the increased oxygen uptake of the bacteria in the presence of the substrate," while "the *rate* of oxidation has been expressed as the metabolic quotient," that is to say—cmm. oxygen absorbed by 1 mgm. in 1 hour.

Results are given in some detail. "Oxidative activity towards the pentoses is considerably less than that towards the hexoses . . . . The amino-acids are oxidized . . . at rates which are in general lower than those of carbohydrates . . . . The biologically important organic acids tested are oxidized at rates which are in general higher than those of amino-acids but lower than those of sugars."

One may naturally ask how closely this oxidative activity is correlated with fermentative activity. The correlation should be high and in the case of the plague bacillus a general parallel between them has been demonstrated.

Glucose and lactic acid have been found to be the best and cheapest carbon sources for addition to media. Among the amino-acids only a certain number are of service as nitrogen and energy sources: serine, alanine, proline, cystine, glutamic acid, glycine, phenylalanine, tyrosine and methionine. *W. F. H.*

RAO (M. Sadashiva). **Further Studies on the Nutrition of the Plague Bacillus: the Rôle of Haematin and Other Compounds.**—*Indian Jl. Med. Res.* 1940. Apr. Vol. 27. No. 4. pp. 833–846. [30 refs.]

Knowledge of bacterial nutritional requirements has advanced greatly in recent years and the study of these simple cells has contributed greatly to the elucidation of problems of metabolism generally. A bacterial growth factor, says the author, "is distinguished from the numerous other compounds that a micro-organism may utilize . . . by the fact that in its absence no growth occurs." The indispensable bacterial growth factor represents already synthesized molecular structure necessary for the nutrition of the organismal cell. "There are two types of bacterial growth factors: (1) simpler molecules, such as the amino acids, and (2) more complex molecules such as thiamin, nicotinic acid, etc." The importance of the complex elements is due to the discovery that they are fundamental in cellular respiration. Many of them are identical with animal vitamins, owing their importance to their activity as co-enzymes in enzymatic oxidation systems. The author has already shown [this *Bulletin*, 1939, Vol. 36, p. 963] that only 3 amino acids—proline, phenylalanine and cystin—are indispensable growth factors for the plague bacillus and that the more complex factors are not essential. The object of the present paper is to show that complex factors may not be indispensable but "possess a *stimulatory* activity on the metabolism and the growth of the organism." As a test of this stimulation observations were made on the ability of a compound added to an amino-acid mixture containing "all the factors essential for the growth of the organism . . . to shorten the lag period of growth, or, in other words, to initiate an earlier multiplication of the added inoculum over that of the control." Six compounds known to function as growth factors or as coenzymes were used: (1) haematin, identical with the X-factor for *H. influenzae*, an activity which is associated with peroxidase and catalase activity, (2) cozymase

or diphosphopyridine nucleotide, identical probably with the V-factor for *H. influenzae*, which by being alternately reduced and oxidized serves to carry hydrogen from and to substrate molecules, (3) thiamin or aneurin, identical with vitamin B_1, (4) nicotinic acid, the anti-pellagra vitamin, (5) isoalloxazine-adenine-dinucleotide, and (6) β-alanine. The standard medium used, of known chemical composition, contained 12 amino acids: its mineral composition was provided from bacterial ash and it was buffered with M/30 phosphate at *p*H 7·4.

The results of this research were :—(1) haematin is highly active in reducing lag in the growth of the plague bacillus, while cozymase, thiamin and nicotinic acid are somewhat less active. "Haematin, thiamin and nicotinic acid together, have the greatest effect recorded." (2) "Haematin, cozymase, thiamin and nicotinic acid stimulate the respiration (oxygen uptake) of actively-growing cultures of the bacillus." (3) "In a study of the effect of the compounds on the oxidation of a few of the more important substrates by washed suspensions of the organism it has been shown that haematin has no stimulatory effect on any of the oxidations studied, but increases the blank O_2-uptake of the suspension. Cozymase stimulates the oxidation of glucose and alanine and the blank O_2-uptake. Thiamin and nicotinic acid stimulate the oxidation of glucose only."

An important suggestion is made that the four growth stimulants—haematin, cozymase, thiamin and nicotinic acid—"may be essential components of the cell, being synthesized in the course of growth, and that their occurrence and ready availability in the environment will greatly facilitate the rapidity of growth and of invasion of the organism." *W. F. H.*

WATS (R. C.) & PUDUVAL (T. K.). **A Study of Some Virulent and Avirulent Strains of** *Pasteurella pestis.—Indian Jl. Med. Res.* 1940. Apr. Vol. 27. No. 4. pp. 823–831. With 2 plates.

In this study of virulent and avirulent strains of plague the subjects dealt with are those of (1) the dissociation forms of the bacillus and its colonies, (2) its "envelope" or "soluble substance." Preliminary study with virulent and avirulent strains, 120/5H, showed that two types of colony were obtained from subcultures on blood agar: "uniform small translucent dew-drop-like colonies with a shiny pearl-like appearance"—designated as "smooth" from the virulent strain, and "large-sized colonies with a pin-pricked sometimes rugose surface and irregular, broken or serrated margins" from the avirulent strain. The smooth colonies were composed of uniform oblong bacilli and the rough colonies of long forms of varying size. No difference was detected in the carbohydrate and protein reactions of virulent and avirulent strains. In broth culture the virulent growth "was uniformly turbid while the avirulent one gave a granular deposit at the bottom and sides with a comparatively clear supernatant fluid."

Perhaps the most interesting observations relate to the "envelope" substance of the bacilli, in the form of soluble substance (S.S.) obtained in the washings of growth, especially at 37°C. It has been very directly connected with the antigenic value of plague vaccines and with the growth of the organism at 27°C. and 37°C., respectively. The present authors' findings are :—(1) "The presence or absence of the S.S. is obviously not dependent on the production of the 'smooth' or 'rough' colonies of a strain, but as a general rule it may be stated

that a strain producing majority of 'rough' colonies is likely to be poor in the S.S. value"; (2) "the presence or absence of the S.S. has no relation to the virulence or non-virulence of the strains studied"; (3) "the titre of the soluble substance seems to indicate the antigenic value of a strain." *W. F. H.*

DEVIGNAT (R.). Diagnostic collectif de la peste murine par la récolte collective des moelles fémorales de rats en eau physiologique et l'inoculation au cobaye de l'émulsion ainsi obtenue. [**Rat Plague Diagnosis by Pooled Marrow Inoculation of Guineapigs.**]—*Ann. Soc. Belge de Méd. Trop.* 1940. Mar. 31. Vol. 20. No. 1. pp. 41–50.

Instead of making cultures of bone marrow the method of subcutaneous inoculation has been adopted and this has rendered it possible to examine 287,962 adult *Mastomys ugandae* since January 1937. The Mastomys is the domestic rat of the Lake Albert region. The procedure is simple and consists in cutting across the leg, just above the knee, plunging a short-bevel needle into the exposed marrow, aspirating and then suspending, by aspiration and re-aspiration, the tissue obtained in 5 cc. sterile physiological salt solution. Young Mastomys rats are rejected. Deratization squads tour their districts once a month, visiting all huts and inoculating daily one guineapig with the bone marrow from the morning's catch of rodents. The same procedure may be adopted for sylvatic species or, if bone marrow cannot be procured, spleens are used and are rubbed into the shaved skin of a guineapig one by one. With this procedure 21 strains of rat plague bacilli were obtained and the plague endemic area of Lake Albert has been defined. Some rats, apparently healthy, were found plague-infected in villages where no human cases of plague had been reported. Those people who had been vaccinated against plague remained free of the disease. *W. F. H.*

LEWILLON (R.), DEVIGNAT (R.) & SCHOETTER (M.). Un cas de méningite pesteuse primitive. [**A Case of Primary Plague Meningitis.**]—*Ann. Soc. Belge de Méd. Trop.* 1940. Mar. 31. Vol. 20. No. 1. pp. 79–82.

A child of nine years was admitted to hospital with a temperature of 37°C. and symptoms of meningitis on the 4th day of illness. Malaria was excluded. The cerebrospinal fluid was purulent but showed no organisms microscopically. The fluid was sown on ascitic agar and blood agar and isolated colonies, which proved to be plague, grew on the former medium. The child died on the 8th day. Puncture tissue, obtained from the heart, liver and lungs, rubbed on the shaven guineapig did not produce plague infection. The conclusion drawn is that the plague infection had not extended beyond the meninges.

W. F. H.

SOKHEY (S. S.) & DIKSHIT (B. B.). **Sulphathiazole in Bubonic Plague.**—*Lancet.* 1940. June 8. pp. 1040–1042. [12 refs.]

The highly standardized methods, which have been worked out in the Haffkine laboratory, Bombay, render experiments easy to set up for the trial of any new form of plague therapy. These experiments

give a clear picture of the relative merits of sulphathiazole and sulphapyridine. Mice bred in the laboratory, 2½ to 4 months old and 21–30 gm. weight, were used with a strain of *P. pestis,* which on subcutaneous injection of 6–12 organisms produced a mortality in the mice of not less than 90 per cent. The drugs were introduced into the stomach in dosages from 10 mgm. a day to 40 mgm. twice a day for 10 days, and in these experiments were given either: (1) at the same time as the infection, or (2) 24, 48, 72 hours after infection. This series may be divided into two groups: (1) before septicaemia has occurred, which includes the administration of the test drug at the same time and 24 hours after infection, and (2) 48 hours and 72 hours after infection, by which time septicaemia is invariable in these mice. In the first of these groups, that is before septicaemia had set in, "as small a dose of sulphathiazole as 10 mgm. a day twice a day for 10 days saved nearly 80 per cent. of the infected animals." In the second group of septicaemic animals "a dose of 40 mgm. twice a day for ten days cures 80–90 per cent. of infected mice." It is shown in these experiments that sulphathiazole is much superior to sulphapyridine in plague and as plague is much more severe in the mouse than in man they hold out a prospect of great advance in the therapy of plague. The authors propose to try out sulphathiazole in the field soon. *W. F. H.*

MOREAU (P.). Deuz cas de peste bubonique traités par l'association sérothérapie E.V. et bactéramide *per os.* [**Successful Treatment of Plague by E.V. Serum and Bacteramide.**]—*Bull. Soc. Path. Exot.* 1940. Apr. 10. Vol. 33. No. 4. pp. 289–292.

Two cases of bubonic plague were proved bacteriologically and were treated by subcutaneous injection of E.V. serum and the sulphonamide preparation "bacteramide" by the mouth. One patient received a total of 250 cc. serum in 3 days and 26 half-gramme tablets of bacteramide in 9 days, the other 260 cc. serum in 3 days and 32 tablets in 10 days. Both patients recovered. *W. F. H.*

GIRARD (G.). Foyers persistants de peste murine à Tananarive. [**Persistent Foci of Rat Plague in Antananarivo.**]—*Bull. Soc. Path. Exot.* 1940. Mar. 13. Vol. 33. No. 3. pp. 209–211.

The subject of this article is the duration of rat plague in restricted areas and the recurrence of active plague in those areas for several years. One example of such persistence of infection occurred on an estate in the country near Antananarivo where a native died of plague in 1927. There was a high mortality among rats at the time. Again, in 1932, a child died of plague and again there was an epizootic among rats and mice. In 1934 there was an epizootic without human cases, and, lastly, in 1939 news was brought of dying rats. This mortality in rats was proved to be due to plague by the inoculation into guineapig and mouse of *X. cheopis* fleas from the dust of the habitations in which dead rats had been found. Other examples are given. The destruction of fleas in huts where the floor is of beaten earth and the roof of thatch is not a practical proposition. From this the deduction is made that efficient vaccination of the population is the best measure for its protection against plague. *W. F. H.*

GIRARD (G.) & RADAODY-RALAROSY (P.). Sort de deux souches de bacilles pesteux atténués inoculées au cobaye par voie sous-cutanée. [**Fate of Two Strains of Attenuated Plague Bacilli Injected Subcutaneously.**]—*C. R. Soc. Biol.* 1940. Vol. 133. No. 4. pp. 580–582.

These experiments were designed to determine how long the living bacilli of the avirulent E.V. strain of the plague bacillus, which is used for vaccination, remained alive in the body of the guineapig and in what organs after subcutaneous injection. A suspension of the organisms, in dose of 1,000 million, was injected 24 hours after preparation. Plague bacilli could only be demonstrated in the blood about 40 hours later and were absent beyond the 2nd day. The spleen was the first of the organs to show invasion towards the 4th day and not beyond the 11th day. The invasion of the liver was somewhat later, but here bacilli persisted for some 13 days. They were only once found in a lymph node and never in the bone marrow, while they were not isolated in culture from the lungs because of overgrowth of associated organisms.

A similar experiment with the avirulent strain Pecha, which is non-immunizing, showed that it does not appear in the blood at all, nor in any of the organs. *W. F. H.*

HAWAII, TERRITORY OF: ANNUAL REPORT OF BOARD OF HEALTH FOR FISCAL YEAR ENDED JUNE 30, 1939. pp. 209–236. With 9 graphs.—**Plague Campaign, Territory of Hawaii.**

This report, or series of reports, is presented by a series of officials and is mainly an administrative record with tables and graphs. It follows the same lines as the report of the previous year [see this *Bulletin*, 1939, Vol. 36, p. 973]. The description of the preparation of banana-phosphorus bait is as follows:—" The phosphorus-banana bait is made by cutting a half ripe banana into segments 1¼ inches in length. Then each segment is cut in half leaving the skin and part of the flesh to act as a hinge; and two holes are then cut in each half of the segment. Into each of these holes is placed a small amount of phosphorus paste. The two sides of the banana segment are then closed to enclose the phosphorus paste and a tooth pick is inserted into the banana segment at an angle to prevent it from splitting open . . . . The bait when made with half-ripe bananas lasts on an average six days under field conditions." *W. F. H.*

AHLUWALIA (C. L.). **Rôle of Calcid Fumigation as a Raticidal and Pulicidal Measure in Anti-Plague Campaign.**—*Indian Med. Gaz.* 1940. Apr. Vol. 75. No. 4. pp. 219–222. With 2 figs.

Cyanide preparations are superior to trapping and baiting operations in the anti-rat plague campaign because they bring about the death not only of rats but also of the equally dangerous fleas. Fumigants such as carbon monoxide and sulphur dioxide gas seem to be less effective, or less convenient than cyanide products. The author has tested the activity of calcium cyanide preparations by means of an artificial rat burrow which permits of a full view of the fumigation operations and, therefore, allows the collection of data on the subject. Two preparations were tested: (1) Cyanogas " A " dust, and (2) Calcid briquettes. The experiments are claimed to show that Calcid is " much

more efficient and economical than cyanogas." Thus 6 gm. Calcid "are at least as efficient as 60 grammes of cyanogas dust." The hydrocyanic acid content of Calcid, however, is only twice that of Cyanogas and it is probable that the greater efficiency of the former is in part due to the type of blower used, which is a combined grinder and blower. It is easy to keep the Calcid, when not being used, in its briquette form and it is a much less expensive preparation than Cyanogas powder because of its greater efficiency. The author concludes with the remarks:—"On account of the dangerous nature of the preparation, calcid has not yet been taken up for use on an extensive scale in many parts of India. With proper control and under trained supervision there is no danger of any kind in operating this valuable product in inhabited areas." In 1935 the number of houses fumigated and number of rat holes closed were 66,357 and 881,974 respectively.

W. F. H.

FEDERATED MALAY STATES. ANNUAL REPORT OF THE INSTITUTE FOR MEDICAL RESEARCH FOR THE YEAR 1938.—**Rat Virus Enquiry.** pp. 140–144 [PARANJOTHY (J. T.)].

Certain bacteria develop powerful exotoxins. Some of these toxins were administered by stomach tube to rats, but little evidence of toxaemia was shown in the case of *Bact. dysenteriae* Shiga toxin, toxin of *S. enteritidis* "Liverpool" and *Cl. botulinum* toxin, even in rather large dose. Non-commercial chemical poison baits recommended by the Department of Agriculture were also experimented with. It was found that with the four which were used, thallium sulphate, sodium arsenite, arsenious oxide and barium carbonate, of concentrations in the bait 0·4, 10, 2 and 20 per cent. respectively, the total deaths out of 45 rats were 22, 31, 31 and 37 respectively. "Rats had to be starved for one or two days before they would ingest completely the doses of bait supplied." The cost per fatal dose (including labour) of these four mentioned poisons was 0·14, 0·054, 0·052 and 0·054 cent, which would make arsenic and barium carbonate the cheapest of the various poison baits used. A dose of 2 gm. poison proved fatal.

W. F. H.

PARANJOTHY (J. T.). **Rat Virus Enquiry Report including a Note on the Raticidal Value of Certain Commercial and Other Chemical Poisons.**—*Bull. Inst. Med. Res. Federated Malay States.* 1939. No. 1. 23 pp.

The plague of the rat is one of medical, industrial and agricultural importance and much annual loss to the community is caused by the depredations and transmitted infections of this rodent. An enquiry of wide scope has been conducted by the author into means, especially of rat virus and poison type, to cope with this menace. "Herd experiments have been undertaken to assess the value of 'viruses' and in some of them a high mortality among rats has been attained." Nevertheless it would seem that the herd experiments performed with accredited strains of Salmonella and Pasteurella have failed in their object of precipitating an epidemic among the rat population. This is the real measure of their effectiveness and it appears that they have been ineffective. It has been the same with Ectromelia virus, which is a filterable virus and pathogenic to mice.

Rats may indirectly be the cause of much sickness among the labour populations of Malayan estates and are the recognized reservoirs of such diseases, for example, as tropical typhus, leptospirosis, melioidosis and plague. With this in view a considerable number of dead rats were examined for infections—rats either found dead or sacrificed in the experiments performed. Out of 74 dead rats found there was no obvious cause of death in 45; pulmonary abscesses like those of melioidosis were found in 17, but *Pf. whitmori* was not isolated although other organisms were grown in culture and helminthic ova and larvae resembling *Strongyloides ratti* were found. Other findings in the blood of sacrificed rats were *Trypanosoma lewisi*, *Bartonella muris ratti* and *Spirillum minus*.

Lastly, the raticidal value of certain commercial viruses and chemical poisons was tested:—Liverpool virus; new Liverpool virus; raxon; ratinin; ratin; vexterm rat biscuits; phosphorus paste; ratbane and certain recommended formulae for baits. *W. F. H.*

D'AMATO (Hugo J.). Lucha contra la rata. [**The Struggle against the Rat.**]—*Bol. Sanitario.* Buenos Aires. 1939. June. Vol. 3. No. 6. pp. 439–442.

JUNIOR (Favorino Prado). O servico de laboratorio na prophylaxia da peste. [**Laboratory Procedure in the Prophylaxis of Plague.**]—*Brasil-Medico.* 1940. Jan. 27. Vol. 54. No. 4. pp. 49–55. With 6 figs.

Plague infection has remained a constant menace in São Paulo after many years of sanitary measures. The necessity of a plague laboratory has fully established itself and an account is given here of the detail and the diagnosis of plague infection in rats, the determination of the various indices in connexion with plague and the biological data regarding rats which are so important in estimating the probabilities of plague epidemic. Month by month the data are recorded of the species of rats (*R. norvegicus*, *R. rattus*, *M. musculus*), numbers of males and females, numbers pregnant, numbers of foetuses, and numbers infected with plague. Care is taken over the separation of *P. pestis* infection from that of *P. pseudotuberculosis rodentium*. The plague bacillus ferments glycogen and is non-motile while the other ferments rhamnose and is motile. *W. F. H.*

BLACKWATER FEVER.

PRÉCIS OF ABSTRACTS IN THIS SECTION.

By injecting haemoglobin intravenously into man, FAIRLEY (p. 833) has found that the renal threshold is between 37 and 102 mgm. per 100 cc.; in amounts greater than these haemoglobinuria was produced, but methaemalbumin could not be detected in the plasma. Increase of bilirubinaemia was found in two of the three subjects. Methaemalbumin was, however, produced in the plasma of patients who received incompatible blood and it is suggested that haemoglobin disintegrating in the circulation is split into globin and haematin (ferrous), the

latter being immediately oxidized to haematin (ferric) which combines with serum albumin to form methaemalbumin. Otherwise, extracorpuscular haemoglobin may be excreted by the kidneys or absorbed by the R.E. system with the production of bilirubin and haemosiderin.

FAIRLEY and MURGATROYD (p. 834) report a case of malaria in which the administration of quinine constantly produced mild attacks of blackwater fever ; the patient was infected with both *P. falciparum* and *P. vivax*. Later, when the malaria was apparently cured, quinine entirely failed to produce haemoglobinuria and there was no evidence of cutaneous sensitivity to quinine. Methaemalbumin was constantly found in the plasma during the attacks of blackwater.

GOTTEN and MACGOWAN (p. 836) report a case of blackwater fever caused by the bite of a spider in America. The patient was a child of 3 who recovered after blood transfusion and injection of 10 per cent. dextrose solution.

CHO (p. 836) describes a case in which death followed the onset of anuria.

PERRIS (p. 837) describes a case of blackwater fever.

MEASHAM (p. 837) writes of the value of *Vitex peduncularis* in the treatment of blackwater fever. CIAVALDINI (p. 837) uses daily intravenous isotonic saline to which quinine and horse serum are added. Details of the method and of the additional measures are given.

C. W.

FAIRLEY (N. Hamilton). **The Fate of Extracorpuscular Circulating Haemoglobin.**—*Brit. Med. Jl.* 1940. Aug. 17. pp. 213–217. With 1 graph. [13 refs.]

In this paper the author describes certain observations which throw some light on the fate of extracorpuscular circulating haemoglobin. The biological relationship of haemoglobin to bilirubin and its subsequent excretion as bile pigment have been established, but the relationship between haemoglobin and haematin is less well understood.

DUESBERG, in an attempt to explain the significance of haematin, as revealed by Schumm's test, in the serum of cases of pernicious anaemia, injected haemoglobin as well as haematin solutions intravenously into man. He found that after the injection of 3 to 6 gm. of haemoglobin intravenously Schumm's test was negative, except in patients with certain diseases of the liver. As these results are not in conformity with certain recent biochemical findings in the haemoglobinurias and haemolytic anaemias, Fairley decided to repeat Duesberg's experiments, injecting larger quantities of haemoglobin and making quantitative determinations of the resulting haemoglobinaemia, as well as serial van den Bergh and Schumm tests.

The technique employed in making Schumm's test was as follows:—

"A given volume of plasma was covered with a layer of ether ; one-tenth the volume of concentrated ammonium sulphide (analar) was then run in with a pipette and subsequently mixed by shaking. A positive reaction is indicated by the appearance of a haemochromogen with a sharply defined α band at 558 mμ in a depth up to 4 cm. of plasma. The presence of much haemoglobin adds to the difficulty of performing the test, since reduced haemoglobin is formed which obscures the haemochromogen bands."

Fairley injected haemoglobin solution into three patients, the amount given varying from 180 mgm. to 350 mgm. per kilo. The maximal

concentration of haemoglobin at the end of the injections varied from 208 to 230 mgm. per 100 cc. The fall in haemoglobinaemia titre was rapid, but it persisted in sufficient concentration to give rise to haemoglobinuria lasting from 7 to 16 hours. The quantity of haemoglobin present in the plasma at the termination of the haemoglobinuria varied from 37 to 69 mgm. The renal threshold for haemoglobin lay somewhere between 37 and 102 mgm. per 100 cc.

The bilirubin estimations were not considered satisfactory owing to technical difficulties, but quite definite and sustained increases were observed in two cases.

In contradistinction to Duesberg's findings, the plasma of all three cases gave a positive Schumm's reaction within 4 to 10 hours, and the reaction persisted for 27 to 34 hours. In all three cases the plasma was examined with a reversion spectroscope for the presence of methaemalbumin, with negative results.

The author next records certain observations made on two cases of incompatible transfusion, and on man and monkeys after intravenous injection of alkaline haematin (ferric).

The following summary is given :—

" Following the intravenous injection of 14 to 25·4 grammes of human haemoglobin in man a positive Schumm reaction was invariably demonstrated in the plasma within four to ten hours, and persisted for twenty-four hours or longer.

" A transient increase in bilirubin was also noted in the plasma in two of the three cases receiving haemoglobin injections.

" Though methaemalbumin was not found spectroscopically following the injection of haemoglobin in these amounts, it was demonstrated in the plasma of two patients who received by mistake incompatible blood containing approximately 45 and 90 grammes of haemoglobin respectively.

" In incompatible transfusion and the other intravascular haemolyses in man, methaemalbumin and not methaemoglobin is found in the plasma ; a mixture of the two pigments has never been recorded *in vivo*, though they do occur together *in vitro* when a human plasma-haemoglobin mixture is incubated at 37°C.

" Extracorpuscular circulating haemoglobin may be (*a*) excreted by the kidney, (*b*) absorbed by the R.-E. cells with the production of bilirubin and haemosiderin, and (*c*) katabolized in the circulation with the production of methaemalbumin.

" It is suggested that haemoglobin disintegrating in the circulation is first split into globin and reduced haematin (ferrous) ; the latter pigment is immediately oxidized to haematin (ferric), which in man combines with serum albumin to form methaemalbumin." *W. Yorke.*

FAIRLEY (N. Hamilton) & MURGATROYD (F.). **Recurrent Blackwater Fever induced by Quinine.**—*Trans. Roy. Soc. Trop. Med. & Hyg.* 1940. Aug. 16. Vol. 34. No. 2. pp. 187–194. With 1 chart.

The authors state that although the close correlation between the administration of quinine to malarial patients and the onset of blackwater fever has long been recognized, it is not often that a malarial patient is found in whom the attacks of haemoglobinuria induced by quinine are sufficiently mild and sufficiently frequent to enable an investigation to be undertaken both during and between the paroxysms of intravascular haemolysis which constitute the fundamental basis of blackwater fever.

The present note deals with such a case. The patient, a woman aged 49, was admitted to hospital on the 2nd June, 1939, with fever which

had developed three days previously. Her blood contained *P. vivax* and *P. falciparum*. The patient had lived in India and Assam for 20 years, and had suffered from recurrent attacks of malaria from 1921 to 1930, but subsequently had been entirely free from fever until three days before admission to hospital. On admission, the red cell count was 4,400,000 and the haemoglobin value 86 per cent. The urine contained only a trace of urobilin.

The patient was given quinine bihydrochloride, 10 grs., thrice daily, commencing on the 3rd June. This treatment was continued during the next day, but at 6 p.m. the patient developed mild blackwater. She had a temperature of 103°F. ; the urine was dark brown in colour and contained oxyhaemoglobin and some methaemoglobin. Antimalarial treatment was stopped, and a mixture containing potassium citrate and sodium bicarbonate was given. The following day the red cell count was 3,600,000 and the haemoglobin value 76 per cent. There was a positive indirect van den Bergh reaction ; methaemalbumin was demonstrable spectroscopically but only in very thick layers of plasma. No parasites were found in the blood.

After an interval of two days, during which the temperature was normal, quinine bihydrochloride was given in progressively increasing dosage, from 1½ grs. on the 7th June to 30 grs. on the 12th and 13th.

On the 10th June, the patient felt unwell. The next evening she had a temperature of 100°F., and the following evening a temperature of 101·4°F. The blood was negative. On the following day (13th June) she had transient haemoglobinuria, and antimalarial treatment was stopped. The next day the patient was icteric, the red cell count was 3,000,000 and the haemoglobin value 70 per cent. The temperature had returned to normal, and remained so for 38 days.

Antimalarial treatment was recommenced on the 28th June, when plasmoquine, 0·01 gm., was administered thrice daily for 5 days without haemoglobinuria developing. On the 17th July, it was decided to ascertain whether quinine could now be given without inducing haemoglobinuria, and quinine bihydrochloride, 5 grs., was given three times without apparent effect, except that the patient did not feel quite as well as usual. Schumm's test for haematin, which was negative before, became positive the day after the quinine was administered. No quinine was given on the 18th and 19th July, but on the 20th, quinine bihydrochloride, 10 grs., was given at 11 a.m. and 2 p.m. The patient complained of deafness at 3 p.m., and at 5.30 p.m., passed dark red urine. The temperature that evening was 101·4°F. The next day the urine cleared.

On the following day (22nd July), quinine bihydrochloride, 10 grs., was given at 4 a.m. and noon. By 4 p.m. the patient was complaining of slight deafness and shivering, and passed 1½ ounces of dark brown urine containing blood pigment ; the temperature was 101°F. Various examinations of the blood were made before and during these attacks.

The following are the conclusions :—

" (1) The laboratory and clinical data recorded refer to a malarial patient studied during recurrent attacks of mild blackwater fever induced by quinine.

" (2) No evidence of dermal hypersensitivity to quinine was found.

" (3) The capacity of quinine to produce blackwater fever appeared in this case to be directly or indirectly related to persisting malarial infection.

" (4) Several months after apparent cure of the malaria, quinine therapy entirely failed to induce haemoglobinuria.

" (5) During and following the bouts of haemoglobinuria, methaemalbumin was invariably demonstrated in the plasma either spectroscopically or by means of Schumm's test.

" (6) Prehaemolytic swelling of the corpuscles associated with spherocytosis and probably increased fragility to hypotonic saline solution were observed.

" (7) The findings suggest that the lytic agent acts directly on the circulating corpuscles, and are compatible with the view that a lytic enzyme or biological haemolysin was implicated."

W. Y.

GOTTEN (Henry B.) & MACGOWAN (J. J.). **Blackwater Fever (Hemoglobinuria) caused by Spider Bite.**—*Jl. Amer. Med. Assoc.* 1940. Apr. 20. Vol. 114. No. 16. p. 1547.

In this note the authors record a case of blackwater fever caused by a spider bite. The spider was probably *Amaurobius ferox*.

The patient was a girl, aged 3, whose family lived in a rural community. The child was bitten on the left flank by a large brown spider. At the time she was seen by the authors she had a temperature of 102°F. and was vomiting frequently. There was a large, swollen, indurated area on the left side just above the buttock, where the spider had punctured the skin. The child did not at the time appear to be extremely ill. The following morning, however, she passed blackwater. The pigment was identified as methaemoglobin, and was present in concentrated amounts. The temperature was still 102°F., the red cell count was 2,940,000, and the haemoglobin level 59 per cent. The leucocyte count was 17,800, with 91 per cent. polymorphonuclear leucocytes, 8 per cent. small lymphocytes and 1 per cent. large mononuclears.

The next morning the colour of the urine had improved, the vomiting had ceased, and the patient was able to take some fluids. During the afternoon, approximately 72 hours after the bite, the child suddenly collapsed. The temperature was 104°F., the pulse rate 160 and the volume very poor, and the respiration 36; she perspired profusely and became cyanotic.

Transfusion of 300 cc. of citrated blood was administered, and this was followed by an injection of 300 cc. of 10 per cent. dextrose. The following morning the temperature was normal and the general condition greatly improved. Convalescence was rapid. *W. Y.*

CHO (Ryoten). Ein Fall von Anurie infolge des Schwarzwasserfiebers. [**Anuria resulting from Blackwater Fever.**]—*Taiwan Igakkai Zassi* (*Jl. Med. Assoc. Formosa*). 1940. July. Vol. 39. No. 7. [In Japanese pp. 973–977. German summary p. 978.]

The onset of anuria in blackwater fever is a sign of grave prognostic importance and death usually follows. The author records such a case in a Japanese, aged 63, who had for 20 years lived in a malarious part of Formosa, but who gave no history of malaria until the present attack, which took the form of tertian fever, for which he was given quinine. On the third day there was diminution of the flow of urine and the urine was dark brown in colour. The diagnosis of blackwater fever was made, and three days later there was anuria. Examination showed severe anaemia with jaundice, weak and rapid pulse and cardiac murmurs, and a spleen just palpable. Erythrocytes numbered

1,160,000, leucocytes 22,300, haemoglobin 40 per cent.; no malaria parasites were found.

Intravenous injection of 5 per cent. sodium bicarbonate was given and 40 per cent. urotropin solution was administered: the patient died. *C. W.*

PERRIS (Emmanuel P.). Ein seltener Fall von Schwarzwasserfieber. [**A Rare Case of Blackwater Fever.**]—*Arch. f. Schiffs- u. Trop.-Hyg.* 1939. Sept. Vol. 43. No. 9. pp. 411–416. [16 refs.]

This note gives in great detail the clinical history of a case of blackwater fever which in the author's opinion presented certain unusual features.

The patient, who had for some time suffered from malaria, was an alcoholic. The history records that he had an attack of malaria in August 1936, and was treated by several courses of atebrin, quinine and plasmoquine. He was reinfected the following May, and again treated with a combination of these drugs.

On the 17th August, 1937, the patient turned up again with malaria and was given an intramuscular injection of 1 gm. of quinine and told to continue with the usual atebrin, quinine and plasmoquine course. A few hours later, however, he had a rigor and passed blackwater. The clinical history of the case from this point until 28th August, when convalescence definitely set in, is given day by day.

To the reviewer there is nothing unusual about the case, apart from the treatment, which was in truth manifold and varied. The case appears to belong to that group characterized by a long series of relatively mild haemolytic attacks. Such cases were recorded by BARRATT and YORKE (1909) and have frequently been reported since. *W. Y.*

MEASHAM (J. E.). *Vitex peduncularis* **in the Treatment of Blackwater Fever.**—*Indian Med. Gaz.* 1940. Jan. Vol. 75. No. 1. pp. 25–28.

The author has tested *Vitex peduncularis* in the treatment of blackwater fever. He administered the drug to a series of eleven cases which occurred in the Anamallai Hills of the South-Western Ghats, between 1936 and 1939. Brief details of the cases are summarized in a table, from which it appears that ten patients recovered and one died. The author concludes that the drug is of definite value in blackwater fever, and adds that he hopes further clinical and pharmacological observations will be made by other workers. [It is hardly necessary to add that much more evidence must be obtained before one is warranted in drawing any such conclusion. References to this drug may be found in this *Bulletin*, 1921, Vol. 17, p. 306, and 1924, Vol. 21, p. 604. In the latter paper CHOPRA, KNOWLES and GUPTA found that it had no effect in malaria.] *W. Y.*

CIAVALDINI. L'hémoglobinurie paludéenne. [**Malarial Haemoglobinuria.**]—*Rev. du Paludisme et de Méd. Trop.* Paris. 1940. Mar. 15. Vol. 2. No. 9. pp. 65–67.

During 30 years' practice in Algeria the author has been called upon to treat 41 cases of malarial haemoglobinuria. As the result of this experience he has adopted certain lines of treatment which he describes.

The basis of the treatment is the intravenous injection of isotonic saline, 250 to 500 cc. a day until the urine becomes normal. On the first three days 1·50 gm. of bihydrochloride of quinine are added to the first 125 cc. of saline injected. No further quinine is given for a week after which its administration is repeated if necessary. To the second half of the saline injection is added 10 to 20 cc. of horse serum with the idea of increasing the rate of blood coagulation. Liver symptoms are relieved by intestinal irrigation with cold boiled water, a quarter of a litre four times a day. Diuretic hot or cold drinks are given: alkaline waters and 50 gm. of lactose a day may be added with profit. To combat cardiac collapse injections of ether, caffein or sparteine, camphorated oil, or adrenalin are useful. *Norman White.*

FEVERS OF THE TYPHUS GROUP AND OTHER FEVERS.

Précis of Abstracts in this Section.

Louse- and flea-borne.—In Bombay, Patel (p. 840) reports seven cases in which typhoid was suspected, but in which the Weil-Felix reaction with *Proteus OX19* was positive, with negative Widal and blood culture.

Sharma (p. 840) similarly reports on the results of Weil-Felix reactions performed on patients with fever but negative to tests for typhoid in Bangalore. Fifty-six positive Weil-Felix reactions were found in 3 years, the majority being positive for *Proteus OX19*. The vector of the disease is thought to be the rat flea.

Musser (p. 841) records an increase in the number of cases of endemic typhus in Louisiana. Prevention lies in the control of rats. Pons (p. 841) reports endemic typhus in Porto Rico.

In Constantinople, Celik (p. 841) describes cases of typhus which differ from epidemic typhus and boutonneuse fever. All gave strong reactions with *Proteus OXL* and considerably weaker reactions with *Proteus OX19*, and all lived in rat-infested dwellings.

From work on guineapigs and on human contacts of cases of typhus Ovalle Sánchez (p. 841) concludes that the injection of serum from convalescent patients is a harmless procedure and has some effect in preventing the disease, but that its activity is small if given towards the end of the period of incubation. The serum used originally gave a Weil-Felix reaction at 1/640 titre, but was concentrated, through a collodion sac, until the titre reached 1/15,000.

Bowdoin and Boston (p. 842) discuss the epidemiology and control of endemic typhus in Georgia, U.S.A. Control is largely a matter of rat extermination or exclusion.

Boston (p. 842) shows that the control of murine typhus in America is a public health engineering problem, and indicates the principles of rat extermination and exclusion from buildings.

Tick-borne.—Although strains of Rocky Mountain fever virus isolated in the Eastern United States have hitherto been less virulent for guineapigs than those isolated in the West, Topping and Dyer (p. 843) now report a highly virulent strain recently isolated in Washington and give details of its effects on guineapigs.

HOLLAND (p. 843) gives information concerning *Dermacentor andersoni* in Alberta, where it is thought to be extending. Ground squirrels and hares are probably the hosts for larvae and nymphs, but domestic stock are the principal hosts of the adults. Open, dry country is less infested than small, deep valleys, since the ticks are sensitive to low humidity. Ticks were found on low bushes and various grasses. ROBERTS (p. 844) gives instructions as to the elimination of *Rhipicephalus sanguineus* from dogs and their kennels in Australia. Derris powder or wash should be used for the animals, and kennels should be carefully sprayed with creosote or crude oil, and old bedding should be burned or boiled. [Although this tick has not been associated with human disease in Australia, it is a vector of boutonneuse fever in France and has been suspected in India.]

FLORMAN and HAFKENSCHIEL (p. 845) note a marked reduction in the number of blood platelets in a case of Rocky Mountain fever.

Mite-borne.—KOUWENAAR (p. 846) gives a detailed description of the pathological appearances found in Sumatran mite fever and in scrub typhus. These appearances are the same in both diseases. They differ from those of classical typhus and endemic typhus in that the chief change is a perivascular infiltration only secondarily attacking the intima, whilst in the classical and endemic forms the primary lesion is destruction of the intima and media followed by perivascular infiltration. LEWTHWAITE and SAVOOR (p. 847) quote the findings on which they base their conclusion that Sumatran mite fever is not a disease *sui generis*, but is identical with tsutsugamushi fever.

LEWTHWAITE (p. 847) has again isolated a virus identical with stock *Proteus OXK* strains but which was obtained from a guineapig infected with strain E, originally a *Proteus OX19* strain obtained from rats caught on an estate where tsutsugamushi fever was present. Details of the findings are given.

Vaccination.—MURGATROYD (p. 848) has summarized present knowledge of immunization against human rickettsial diseases. In discussion of this paper FINDLAY points out that in N. Africa the natives possess a certain degree of inherited or acquired immunity and that the reactions to vaccination are slight, but that, in Europeans, living vaccines of the murine type give rise to severe reactions. Killed vaccines confer a certain amount of immunity.

DURAND and SPARROW (p. 849) show that if the viruses of typhus and boutonneuse fever are administered to mice by the nasal route there results haemorrhagic pneumonia in which large numbers of Rickettsia are found. DURAND *et al.* (p. 849) also found that the virus of Rocky Mountain fever inoculated by the nasal route in mice, rabbits and squirrels gave haemorrhagic pneumonia rich in Rickettsia. DURAND and GIROUD (p. 849) have used Rickettsiae from the lungs of infected mice, emulsified in formalin, as a vaccine in guineapigs with success. In man and monkeys inoculations of this vaccine produced positive Weil-Felix reactions. They (p. 850) obtained success with a similar vaccine of Rocky Mountain fever virus, in experiments with guineapigs.

SPARROW and MARESCHAL (p. 850) infected men with a murine virus contained in the intestines of inoculated lice, by instillation of an emulsion into the conjunctival sac. Mild fever resulted but no virus could be found in the blood. The men were later found to be immune to inoculations of classical and murine typhus.

LAIGRET (p. 850) has experimented with a method of vaccination against typhus by application of a vaccine to the scarified skin.

Other fevers.—DERRICK and SMITH (p. 851) have found a bandicoot, *Isoodon torosus*, naturally infected with the virus of Q fever in Australia. *Haemaphysalis humerosa* is known to be a natural vector of this disease in animals, and SMITH (p. 851) has investigated its importance in producing infection in man. The faeces of infected ticks are highly infective and are capable of infecting guineapigs even through the unabraded skin. This would appear to be the natural mode of infection of vertebrates, and the infection of abattoir workers may be attributed to this.

COX (p. 852) sums up recent information on *Rickettsia diaporica* and the disease caused by it, which it is proposed to name American Q fever on the grounds that it is identical with Australian Q fever.

HERTIG (p. 853) has cultivated *Bartonella bacilliformis* on semi-solid Noguchi leptospira medium.

MAYRAC (p. 853) describes an outbreak of three-day fever in New Caledonia.

SHORTT *et al.* (p. 853) show that monkeys are susceptible to infection with the virus of sandfly fever and that the virus can be passaged through mice ; neutralizing bodies can be demonstrated. In man the virus can be recovered up to 3 or 4 weeks after the onset of fever, and can be cultured on the chorio-allantoic membrane of the fowl embryo. The cultured virus used as a vaccine appeared to produce some degree of immunity in man. *C. W.*

PATEL (T. B.). **Typhus Fever in Bombay.**—*Indian Med. Gaz.* 1940. Apr. Vol. 75. No. 4. pp. 208–209.

In the Fever Hospital cases which had been admitted as possibly typhoid and which gave negative Widal reactions and blood cultures were tested for the Weil-Felix reaction. Seven of these gave positive reactions with *Proteus OX19* and are described in the paper ; all recovered. *D. Harvey.*

SHARMA (L. R.). **"Non-Epidemic Typhus" Fever in the Civil Population of Bangalore (Civil and Military Station).**—*Indian Med. Gaz.* 1940. July. Vol. 75. No. 7. pp. 398–401. With 2 charts. [10 refs.]

In the 3 years 1936, 1937 and 1938 all cases of fever which had not given a positive Widal reaction or a positive blood culture were also tested for the Weil-Felix reaction ; as a result some 56 cases with positive reactions were found : 50 for *Proteus OX19*, 5 with *OX2* and one, an imported case, for *OXK*. In the majority of the cases the fever lasted for 10 to 18 days and typical rashes were seen in 12 cases ; all the patients recovered.

The vector of endemic typhus in Bangalore has not been determined, but the majority of the cases occurred among poor people in the city and therefore the rat flea is probably responsible. In one case, however, there was a history of tick bite when the patient was on a picnic party in the jungle. *D. H.*

MUSSER (J. H.). **Typhus Fever in Louisiana.**—*New Orleans Med. & Surg. Jl.* 1940. July. Vol. 93. No. 1. pp. 39–42.

Cases were first reported in the State in 1929 and 115 cases were notified in 1939.

The typhus is entirely of the endemic type. It is noted that this disease is a disease of the Southern States and the reasons given for this are, the milder climate, the fact that houses are not strongly built, and are not rat proof, and that ample food supply is readily available for rats.

Prevention lies in control of rats. *D. H.*

EVANS (Earl F.). **Endemic Typhus of the Southeastern States.**—*U.S. Nav. Med. Bull.* 1940. Apr. Vol. 38. No. 2. pp. 210–218. [16 refs.]

A general survey of the epidemiology and clinical aspects of the disease with descriptions of two cases seen by the author. *D. H.*

PONS (Juan A.). **Is there Brill's Disease in Puerto Rico ?**—*Bol. Asoc. Med. de Puerto Rico.* 1940. June. Vol. 32. No. 6. pp. 196–201.

The term Brill's disease is used in this paper as a synonym for endemic typhus and the answer to the query in the title is in the affirmative.

Eight cases are described, 3 of which gave a strongly positive Weil-Felix reaction and in 6 a typical rash was present. *D. H.*

CELIK (O. Serefettin). Quelques cas atypiques de fièvre exanthématique à Istanbul. [**Some Atypical Cases of Typhus Fever in Constantinople.**]—*Bull. Soc. Path. Exot.* 1940. Apr. 10. Vol. 33. No. 4. pp. 260–265.

In the last 2 years some 18 cases have been met with which differed clinically and serologically from classical typhus and boutonneuse fever. Four cases are described in detail. There was no primary sore noted on any of the patients and the rash differed from that of true typhus or boutonneuse fever. Serologically all the cases showed a strong positive reaction for the Lima strain of *Proteus OXL* and considerably less for *OX19*. All the people lived in rat-infested dwellings. *D. H.*

OVALLE SÁNCHEZ (Héctor). Estudios sobre tifo exantematico. Suero de convalecientes concentrado. Nota preliminar. [**Concentrated Serum of Convalescents in the Treatment of Typhus.**]—*Rev. Chilena de Hig. y Med. Preventiva.* 1939. July–Dec. Vol. 2. No. 7–12. pp. 349–361. With 4 figs. & 16 charts. [12 refs.]

A serum with a Weil-Felix titre of 1/640 was concentrated by passage through a collodion sac which allowed water and salts to pass, but not the globulin antibodies, till a serum of a titre of 1/15,000 was obtained. With this, a series of tests were carried out on 80 guineapigs, divided into 16 groups of 5 and kept under observation for 2 weeks after the experiment was made. The virulent material for intraperitoneal inoculation was cerebral tissue with defibrinated blood diluted with physiological saline. Inoculation of the serum was made from two days before that of the virus to eight days after and the results are depicted in a series of 16 graphs.

Attention was then directed to human subjects ; 50 persons, direct contacts of 12 cases, were injected. Of these 50, two only developed typhus ; one of these inoculated five days before his attack presented a very mild course. The author concludes that injection of the serum of convalescents is a harmless procedure, but that its activity is small if given towards the end of the period of incubation. *H. H. S.*

BOWDOIN (C. D.) & BOSTON (Roy J.). **A Preliminary Report on the Practical Epidemiology and Control of Endemic Typhus Fever in Georgia.**—*Amer. Jl. Trop. Med.* 1940. July. Vol. 20. No. 4. pp. 537–549. With 4 graphs, 2 charts and 1 map.

This paper is complementary to that on the engineering side of the problem which is summarized below.

Statistics are dealt with in the first place : from 1932 to 1939, 4,762 cases of typhus were reported in the State of Georgia. July, August and September are the peak periods of the year. The attack rate in whites is just over 7 times that in the coloured people. Practically all the cases occur in the small towns and villages and these are situated for the most part in the Southern Counties of the State ; this is accounted for by the fact that in this part of the State there is abundant food supply and harbourage for rats. Spot maps of cases have been prepared for these towns and villages and on the information contained in these maps the control measures have been initiated.

The programme is essentially one of :—

1. Epidemiological investigation.
2. Education.
3. Garbage removal and cleaning up of premises.
4. Rat extermination.
5. Rat proofing, which is the final, and of course the only, permanent measure of protection.

It is stressed that epidemiological investigations are essential to the control of any disease and this statement is true of typhus. Control measures cannot be employed efficiently and economically until it is known whence the disease is spreading. *D. H.*

BOSTON (Roy. J.). **Public Health Engineering Phases of Murine Typhus Control.**—*Amer. Jl. Public Health.* 1940. June. Vol. 30. No. 6. pp. 619–626. With 4 figs. & 1 chart.

Because of the increasing incidence and the nature of the problem of murine typhus fever in the State of Georgia a Typhus Control Unit, under the direction of a public health engineer, was established in 1937. As a result of preliminary studies of the State-wide problem the control programme adopted, and now being carried out, consists of preliminary investigations and control measures employing the use of rat extermination, garbage control and cleaning up, and rat proofing. The conclusions are that the control of murine typhus fever is basically a public health engineering problem.

The use of toxic red squill bait in the form of " torpedoes " (½ oz. bait in a roll of twisted paper) is recommended as an effective means of temporarily reducing the rat population ; garbage control and cleaning up affords an effective means of reducing the rat population on premises through starvation and reduction of harbourage. Rat

proofing, however, is the only permanent means of rat control and, therefore, of murine typhus control. This is a long-term policy but much can be done by what is termed " vent stoppage," *i.e.* closing of all openings in the exterior walls of buildings to prevent the ingress and egress of rats, and also the flashing of doors with galvanized sheet iron. *D. H.*

RAYMOND. Épidémiologie et prophylaxie du typhus exanthématique. Maladie No. 2. Conférence de Vulgarisation faite aux Médecins de Réserve de la Région de Paris. [**Epidemiology and Prophylaxis of Typhus.**]—*Rev. Service Santé Milit.* 1940. Mar. Vol. 112. No. 3. pp. 335–347.

TOPPING (Norman H.) & DYER (R. E.). **A Highly Virulent Strain of Rocky Mountain Spotted Fever Virus isolated in the Eastern United States.**—*Public Health Rep.* 1940. Apr. 26. Vol. 55. No. 17. pp. 728–731. With 1 fig.

So far, strains of virus isolated from cases of Rocky Mountain fever occurring in the Eastern States have been less virulent for guineapigs than strains isolated in the West. Also, in cases of the fever in the East the mortality is less than in the Western States. As regards guineapigs infected with Eastern strains the scrotal lesions are rare and when they do occur there is no necrosis or sloughing; the incubation period is longer and the mortality rate is from 7 to 40 per cent. as compared with 80 to 90 per cent. with the western strains. However, a strain of virus (W) recently isolated from a case of fever in the East gave the following reactions as compared with a known virulent strain from a Western case :—

Strain of Virus	Locality	Number of Guineapigs	Died	Recovered	Percent. Mortality
B.R. ...	Montana	50	41	9	82
W	Washington	22	19	3	86
M	Washington	41	14	27	34

Strain of Virus	Guineapigs	Incubation Period, Days	Died	Recovered	Scrotal Lesions
B.R.... ...	12	2·6	7	5	9
W	12	3·1	6	6	10
M	12	4·2	0	12	0

B.R.=Virulent Montana strain.
W.=Recently isolated strain in Washington.
M.=Usual type of Eastern strain from stock. *D. H.*

HOLLAND (G. P.). **Notes on the Ecology of** *Dermacentor andersoni* **in Southern Alberta.**—*Proc. Entom. Soc. B.C.* Victoria, B.C. 1940. No. 36. pp. 8–11. [Summarized in *Rev. Applied Entom.* Ser. B. 1940. Aug. Vol. 28. Pt. 8. p. 143.]

" Since cases of Rocky Mountain spotted fever have occurred in Alberta during recent years, a study of *Dermacentor andersoni*, Stiles,

the vector of this and other diseases, is of importance, and data on its habits and distribution, which are given in this paper, were collected in southern Alberta during April–July 1938. At the present time, *D. andersoni* is known to occur from the southern border of Alberta across the breadth of the Province and north at least to Red Deer (about 52°15′ N. lat.). The general opinion seems to be that the infested areas are becoming increasingly abundant and that the tick is gradually spreading northwards. In the course of a survey along the southern border of the Province, only the short-grass prairie in the south-eastern corner was found to be heavily infested (though there were a few ticks in other localities), and it was in this area that most of the collecting was carried out. Ground squirrels (*Citellus richardsoni*) and hares (*Lepus townsendi campanius*) are fairly abundant there and are probably the more important hosts of the larval and nymphal ticks. The adult ticks are also picked up by the hares and probably by the native antelope (*Antilocapra americana*), which is still fairly common, but domestic sheep, cattle and horses are their more important hosts. Although the ticks were sometimes fairly numerous on the open rangelands, the largest numbers were obtained in the vicinity of water. The old river beds that traverse this part of the country were nearly all infested to a greater or lesser degree, the smaller deeper valleys of tributaries with deeply sloping sides being more favourable habitats than the wide bottomlands of the main river beds. Since ticks are sensitive to lack of humidity, it is not surprising that open, dry and wind-swept areas were less frequented. The ticks occurred on low bushes and various grasses. On dull, warm humid days, especially just prior to rain, they were very active and readily collected on the drags (pieces of white flannelette, one yard square), whereas on cold and windy days or those that were particularly hot and dry, most of them were too lethargic to hang on to the drag or fell off it before they could be transferred to vials. In south-eastern Alberta, the tick season apparently begins late in March, depending on weather conditions, reaches its peak in May and early June and comes to an end in July. Owing to the unusually wet spring of 1938, only about 35 days were suitable for collecting, but during this time nearly 22,000 ticks were obtained."

ROBERTS (F. H. S.). **The Brown Dog Tick** (*Rhipicephalus sanguineus*). —*Queensland Agric. Jl.* Brisbane. 1939. Vol. 52. Pt. 5. pp. 529–530. With 1 fig. [Summarized in *Rev. Applied Entom.* Ser. B. 1940. Aug. Vol. 28. Pt. 8. p. 146.]

"Brief popular notes are given on the morphology and bionomics of *Rhipicephalus sanguineus*, Latr., which breeds very rapidly in the climate of Queensland and causes loss of vitality in dogs on which it is abundant, but is not associated in Australia with the transmission of disease. Occasionally it is carried into dwellings by dogs and, although it seldom attacks man, it may become sufficiently numerous to be considered an important household pest. Arsenical and phenolic dips may kill ticks on the dog, but do not prevent larvae, nymphs and young adults in the animal's sleeping quarters from attaching themselves shortly afterwards. Treatment with derris has been found to be the only way of preventing immediate reinfestation. It may be applied as a powder shaken well into the coat and on to the skin or as a wash

that is allowed to dry on the coat. The wash is made by soaking 2 oz. derris powder overnight in 1 gal. water and adding, just before use next morning, enough soap to make a good lather. Derris should be used with caution on young puppies, especially those of delicate breeds, and care should be taken to keep it away from the animals' eyes, since it may cause them to become inflamed. Treatment should be carried out every 6–7 days until the infestation is eradicated, care being taken to remove by hand any ticks in the ears, on the eyelids or between the toes. All old bedding should be burned or boiled, and the bedding subsequently used should be examined and cleaned every week. All litter round the animal's sleeping quarters should be burned. Kennels and outhouses should be sprayed with creosote oil or crude oil, care being taken to force the spray into cracks and crevices. Houses that are heavily infested should be fumigated."

[*Rhipicephalus sanguineus* is a vector of boutonneuse fever in France and has been suspected in India.]

FLORMAN (A. L.) & HAFKENSCHIEL (J.). **The Eastern Variety of Rocky Mountain Spotted Fever. The Experience in the Adult Medical Service of the Johns Hopkins Hospital, including the Report of a Fatal Case showing Thrombocytopenia.**—*Bull. Johns Hopkins Hosp.* 1940. Feb. Vol. 66. No. 2. pp. 123–133.

In the last nine years six cases of Rocky Mountain fever, of which three proved fatal, have been admitted to the Johns Hopkins Hospital. One of these cases is described in detail with autopsy notes and the other six cases are analysed from the point of view of history and clinical and laboratory findings. A marked reduction of the number of blood platelets was noted in the case described. *D. H.*

OLMER (D.) & OLMER (J.). Fièvre boutonneuse. Ses relations avec les autres Rickettsioses exanthématiques. [**Boutonneuse Fever. Its Relationship with Other Fevers of the Typhus Group.**]—*Marseille-Méd.* 1940. Jan. 15. Vol. 77. No. 1. pp. 9–14. With 1 fig.

A general review of the clinical and epidemiological features of the disease as studied by the authors over a period of 18 years in a district where boutonneuse fever is the only type of typhus-like fever which occurs, and which therefore could not be confused with other members of the group with which it is contrasted. *D. H.*

i. OLMER (D.) & PIERI (Jean). Diagnostic des fièvres exanthématiques. [**Diagnosis of Fevers of the Typhus Group.**]—*Marseille-Méd.* 1940. Jan. 15. Vol. 77. No. 1. pp. 15–17.

ii. —— & ——. Sur les cas de fièvre exanthématique observés en période de guerre. [**Boutonneuse Fever in War Time.**]—*Ibid.* pp. 18–21.

i. In view of war conditions and the possibility of outbreaks of typhus and trench fever the authors review the subject for the benefit of those who have had no experience of the diseases. Already some increase in the number of cases of boutonneuse fever has been noted and all suspicious cases should be isolated and treated.

ii. Cases of boutonneuse fever observed during the war period are described. *D. H.*

KOUWENAAR (W.). Onderzoekingen over Sumatraansche Rickettsiosen. XI. De pathologische anatomie van de mijtekoorts bij den mensch. [**Pathology of Mite-borne Typhus.**]—*Geneesk. Tijdschr. v. Nederl.-Indië.* 1940. Apr. 30. Vol. 80. No. 18. pp. 1119–1140. With 29 figs. on 6 plates. [30 refs.] English summary.

" A description is given of the pathological examination of 23 cases of Sumatran mite fever and 3 cases of scrub typhus (being a disease of the typhus group of fevers, with formation of agglutinins against Proteus X Kingsbury bacilli ; mite fever with and scrub typhus without a primary ulcer).

" *Skin* : mononuclear infiltrations around vessels, glands and nerves, sometimes with infiltration of the media, but never with endovasculitis. The primary ulcer is a coagulation necrosis resulting from the bite of the infecting arthropoda.

" *Lymphatic glands* : swelling, proliferation of the stroma, focal necrosis, catarrh of the sinuses with macrophagia of the endothelial cells.

" *Spleen* : enlargement ; in uncomplicated cases the consistency is firm, but when pneumonia is present a regular acute splenitis may develop. Microscopically the usual picture of acute splenitis is found, with distinct erythrophagia in the endothelial cells. As more specific changes infiltrations in the trabeculae are found, especially around the vessels, with forming of subendothelial granulomata in the venae.

" *Heart* : diffuse acute interstitial myocarditis with infiltrations of mononuclear cells. Perivascular infiltrations with penetration in the wall of the vessels and sometimes destruction of the intima with formation of a thrombus. The muscle cells are only very slightly damaged.

" *Lungs* : in many cases pneumonia is present. In the septa perivascular infiltration may be seen.

" *Stomach and intestine* : no specific lesions were found. There are often petechiae in the mucosa, and once an acute ulcer of the duodenum had developed, without specific microscopical changes in the bottom.

" *Liver* : degeneration and regeneration with mitosis in the liver cells and the KUPFFER cells. Erythrophagia. Small foci of degeneration, necrosis and infiltration.

" *Kidney* : Interstitial infiltrations, especially around the arctiform vessels. Only slight changes of the parenchyma were found.

" *Testes* : interstitial infiltration with mononuclear cells.

" *Brain* : many small foci of infiltration around the smaller vessels, with penetration of the infiltration in the wall, necrosis of the intima, thrombosis and sometimes destruction of the whole vessel.

" These changes give a good explanation of the clinical symptoms : apart from the general infection myocardial and cerebral symptoms are predominant. Death is often caused by heart failure. As the changes in the specific tissue of the different organs are only slight, the disease being located interstitially, no sequelae are found. Only a postinfectious myocarditis may trouble the patient for some months.

" The pathological changes differ from those found in classical epidemic and endemic typhus (with agglutination against Proteus

X 19) as the chief change is a perivascular infiltration, only secondarily attacking the intima, whilst in classical typhus the primary disease is a destruction of the intima and media, followed by perivascular infiltrations."

LEWTHWAITE (R.) & SAVOOR (S. R.). **The Relation of Sumatran Mite Fever to the Tsutsugamushi Disease of British Malaya.**—*Brit. Jl. Experim. Path.* 1940. June. Vol. 21. No. 3. pp. 117–125.

The authors have already conclusively proved that " scrub typhus " and tsutsugamushi fever of Malaya are one and the same disease ; scrub typhus is tsutsugamushi fever without the primary ulcer. A similar disease has been described in Sumatra by Dutch workers who have given it the name of Sumatran mite fever and this could be described as scrub typhus with a primary ulcer ; this disease also belongs to the *Proteus XK* group of typhus-like fevers.

Recently, strains of the virus of this disease, sent by the Dutch authorities in infected white mice, have been received in Malaya. These strains have now been compared with the local strains of tsutsugamushi fever. It was soon found that it was not possible to maintain the strains in white mice, a finding which was contrary to the records from Sumatra. Guineapigs also could not be infected, which is true also with regard to local strains ; rabbits, however, were readily infected by the intraocular route and in a period of 2 years these strains have been passaged from rabbit to rabbit for 70 generations.

The reaction in the eye of the rabbit was typical of that caused by the local virus of tsutsugamushi fever and numerous Rickettsiae were found in Descemet's membrane, these resembled *R. orientalis* ; also the sera of these rabbits gave a positive Weil-Felix reaction with *Proteus OXK*.

A local papular reaction followed inoculation into monkeys and the sera of these animals also gave a positive reaction with *Proteus OXK*.

The results of cross immunity experiments indicated that the strains of virus of Sumatran mite fever and the 3 local strains of tsutsugamushi compared with it are identical.

The results of these experiments were not clear cut in all instances : although the local strains protected against the Sumatra strain, there was only partial protection in animals inoculated with the Sumatra strain. This is explained by the fact that the local strains had been passaged for many generations whereas the Sumatra strain was recently isolated ; it has also been shown that monkeys may lose immunity to tsutsugamushi in a period of months.

The authors conclude that Sumatran mite fever is not a disease *sui generis* but is identical with tsutsugamushi fever. *D. H.*

FEDERATED MALAY STATES. ANNUAL REPORT OF THE INSTITUTE FOR MEDICAL RESEARCH FOR THE YEAR 1938.—[**Typhus-like Fevers.** pp. 114–131. LEWTHWAITE (R.).]

In a previous communication by the same author [this *Bulletin*, 1939, Vol. 36, p. 463] it was noted that from a strain of murine typhus virus, *Proteus X19* type, isolated from rats caught on an estate where tsutsugamushi fever was present, and passaged through guineapigs, a daughter strain was obtained which gave all the reactions in animals

and in serological tests of *Proteus XK* type tsutsugamushi virus. Cross immunity experiments were carried out with known *XK* strains and complete cross immunity was demonstrated; in 8 tests with the original *X19* strain there was no cross immunity; also rabbits which had given the ocular reaction with stock *XK* strains were immune to reinoculation with the new *OXK* strain.

In view of the importance of this finding the whole series of experiments was repeated this year. Fleas were fed on 4 guineapigs infected with the original E strain *OX19*; 3 guineapigs were then placed in the tank with the infected fleas and they became infected. The strain was then passaged by injection of infected brains; among the animals of the third generation killed one was found to give the post mortem signs of tsutsugamushi virus infection, namely peritoneal exudate with ropy secretion but without scrotal reaction; virulent infections were produced in guineapigs infected from this animal. The strain was maintained by passage in guineapigs and also was easily maintained in the eye of rabbits, and the typical reaction was noted with numerous Rickettsiae in the cornea. Cross immunity experiments were again carried out; in 9 tests with the parent *OX19* strain there was no cross immunity, in 8 tests with the stock *OXK* strains immunity was complete. In two rabbits which had been inoculated with the *OXK* stock virus there was no reaction when the eye was inoculated with the freshly isolated *OXK* strain: and *vice versa*.

This new strain is identical with the *OXK* strain of the previous year and both were derived from the same *OX19* strain isolated from wild rats. In this same report the relation of the virus of Sumatran mite fever to that of tsutsugamushi in Malaya is discussed (see LEWTHWAITE and SAVOOR above). *D. H.*

MURGATROYD (Frederick). **A Review of Immunization against Human Rickettsial Diseases.**—*Trans. Roy. Soc. Trop. Med. & Hyg.* 1940. June 27. Vol. 34. No. 1. pp. 1–32. [152 refs.] Discussion pp. 33–36 [FINDLAY (G. M.), LUSH (D.), CHRISTOPHERS (Rickard) & MURGATROYD (F.) (in reply).]

A most comprehensive review of the subject compiled from 152 papers, the majority of which have been summarized from time to time in this *Bulletin* but are now made available in one review.

In the discussion which followed the reading of the paper Dr. Findlay stated that he had recently had the opportunity of seeing something of this work at first hand in North Africa and in Paris. He referred to one or two points in the paper and pointed out that in North Africa the inoculations had been carried out in a native population among whom typhus had been endemic for generations and who therefore possessed a certain amount of acquired or inherited immunity; this immunity was indicated by the fact that in Tunis about one-third of the native inhabitants tested gave a positive Weil-Felix reaction. In these people the reaction to vaccination was slight, but in Europeans the living vaccine of the murine type gave rise to severe reactions and for this reason had been discontinued in some places. Dr. Findlay himself had been inoculated with the Weigl vaccine some time ago and recently with a vaccine prepared from the brains of mice infected with the exanthematic virus killed by formalin; a laboratory infection with the murine virus followed but the attack was mild with only a few days' fever, whereas an unvaccinated fellow worker had a typical

and moderately severe attack with high fever ; it would appear from this that the killed vaccines do confer a certain amount of immunity.

D. H.

DURAND (Paul) & SPARROW (Hélène). Développement dans le poumon des virus typhiques et boutonneux instillés par voie respiratoire. [**Development in the Lung of the Viruses of Typhus and Boutonneuse Fever when instilled by the Respiratory Route.**]—*Arch. Inst. Pasteur de Tunis.* 1940. Mar. Vol. 29. No. 1. pp. 1–24. With 6 figs.

Young white mice are anaesthetized with ether and 4 drops of inoculum of the virus Tunis No. 1 are administered by the nasal route. At first death occurs in 4 to 7 days but when passaged from mouse to mouse death takes place in 48 hours ; at post mortem a condition of haemorrhagic pneumonia is found ; and in the hepatized portions of lung enormous numbers of Rickettsiae are found ; these are also present in the normal parts of lung ; the brain of the mice is also infective but Rickettsiae are not numerous.

If mice are inoculated with the historic typhus virus obtained from infected lice the same condition occurs and enormous numbers of Rickettsiae are found in the lungs of the mice.

If the virus of boutonneuse fever is used and inoculated in the same way a similar type of pneumonia occurs in the mice but the Rickettsiae are not so numerous. The inoculum in this instance was obtained from infected ticks.

D. H.

DURAND (Paul), GIROUD (Paul) & SPARROW (Hélène). Inoculation pulmonaire du virus pourpré (fièvre des Montagnes Rocheuses). [**Pulmonary Inoculation of the Virus of Rocky Mountain Fever.**]—*C. R. Acad. Sci.* 1940. May 27. Vol. 210. No. 22. pp. 751–753.

Mice, rabbits and squirrels were inoculated with the virus of Rocky Mountain fever by the nasal route into the lungs. Three strains of the virus were employed, one highly virulent, one less virulent, and one of only slight virulence.

Exactly the same conditions were produced in these animals with these viruses as were obtained when typhus viruses were instilled by the same route, namely haemorrhagic pneumonia rich in Rickettsiae, in the first instance the inoculum was obtained from infected guineapigs and when an emulsion of infected ticks was employed the same results followed.

D. H.

DURAND (Paul) & GIROUD (Paul). Essais de vaccination contre le typhus historique au moyen de Rickettsias tuées par le formol (souches pulmonaires). [**Attempts at Vaccination against Historic Typhus by means of Rickettsia killed by Formalin.**]—*Arch. Inst. Pasteur de Tunis.* 1940. Mar. Vol. 29. No. 1. pp. 25–52. With 7 figs.

Numerous Rickettsiae obtained from the lungs of infected mice, as reported in the previous paper, were emulsified in formalin serum. Guineapigs were given repeated doses of the vaccine and there was no reaction, but when tested later with large doses of living virus there was solid immunity.

Monkeys and volunteers were inoculated with doses of the vaccine and again there was no reaction, but the sera of the inoculated men and animals gave a positive Weil-Felix reaction and also neutralized the virus. *D. H.*

DURAND (Paul) & GIROUD (Paul). Essais de vaccination contre le virus pourpré (fièvre des Montagnes Rocheuses) au moyen de rickettsies tuées par le formol (souches pulmonaires). [**Attempts at Vaccination against the Virus of Rocky Mountain Fever by means of Rickettsia killed by Formalin.**]—*C. R. Acad. Sci.* 1940. May 27. Vol. 210. No. 22. pp. 753–754.

The vaccine consisted of the emulsified lung of infected mice in formalinized serum and was kept in the refrigerator for 5 days before use.

In guineapigs given 3 doses of 0·5 cc. there was no reaction. Thirty days later they were tested; 7 guineapigs were given as test dose 100 to 1,000 times the minimal infective dose, in one animal only there was slight fever, in the others no reaction either local or general was produced. Five control animals all gave scrotal reactions and 3 died. *D. H.*

SPARROW (H.) & MARESCHAL (P.). Immunisation de l'homme par voie oculaire avec les Rickettsias du virus murin 1 de Tunis. [**Immunization of Man by the Ocular Route with the Rickettsia of the Murine Virus Tunis No. 1.**]—*Arch. Inst. Pasteur de Tunis.* 1940. Mar. Vol. 29. No. 1. pp. 53–65. With 7 charts.

Lice were inoculated with the virus obtained from the brain of infected rats.

Fifteen lice intestines of the 6th passage were taken and emulsified in 1 cc. of sterile saline; 2 drops of this emulsion were placed on the conjunctiva of 7 young men (cases of dementia praecox). All reacted with a mild fever lasting 10 days but there were no other symptoms and no virus was found in the blood of the patients. Six months later the men were tested by repeated eye drops of the same emulsion and no reaction whatever was seen. Seven months later the men proved immune to inoculations with the virus of historic typhus and 20 months later were still immune to the virus of murine typhus. *D. H.*

LAIGRET (J.). A propos de la vaccination contre la fièvre jaune par la voie dermique. Essais de vaccination contre le typhus par la même voie. [**Concerning Vaccination against Yellow Fever through the Skin. Attempts at Vaccination against Typhus by the Same Route.**]—*Bull. Soc. Path. Exot.* 1940. Apr. 10. Vol. 33. No. 4. pp. 227–230.

The method of vaccinating against yellow fever by means of emulsions of the brain of infected mice applied through the scarified skin has been carried out for three or four years with remarkable success [see this *Bulletin*, 1940, Vol. 37, p. 92].

The author is now experimenting with a typhus vaccine prepared from the brain of infected mice and inoculated through the skin and hopes to obtain results similar to those obtained with the yellow fever vaccine. *D. H.*

DERRICK (E. H.) & SMITH (D. J. W.). **Studies in the Epidemiology of Q Fever. 2. The Isolation of Three Strains of** *Rickettsia burneti* **from the Bandicoot** *Isoodon torosus*.—*Australian Jl. Experim. Biol. & Med. Sci.* 1940. June. Vol. 18. Pt. 2. pp. 99–102. With 1 fig. [Summary appears also in *Bulletin of Hygiene*.]

One of 60 bandicoots (*Isoodon torosus*) captured on the mainland of Australia, in the Namour district, was found naturally infected with the virus of Q fever, as were also two of 43 captured on Moreton Island. Guineapigs primarily inoculated from two of these three remained afebrile for ten days, but inoculation of their livers and spleens caused infection of second passage animals. With the third strain the guinea-pig inoculated primarily was infected. All three bandicoot strains gave rise to the characteristic rickettsiae when inoculated into mice, and the sera of all three bandicoots agglutinated *R. burneti*.

The first of the three was harbouring, when arriving at the laboratory, 70 nymphal scrub ticks, *Ixodes holocyclus*, and 11 fleas (Pygiopsylla), but these ectoparasites did not infect guineapigs. The second harboured 3 male ticks, *Haemaphysalis humerosa*, which also failed to infect a guineapig on inoculation. The same species of tick, however, on the third bandicoot was infected.

Briefly, therefore, bandicoots have been found naturally infected with the virus of Q fever, which confirms the previously described agglutination of *R. burneti* by the serum of captured bandicoots [*Bull. of Hyg.*, 1939, Vol. 14, p. 486 ; 1940, Vol. 15, p. 303], and establishes the *Isoodon torosus* as a natural reservoir host of the virus. *H. H. S.*

SMITH (D. J. W.). **Studies in the Epidemiology of Q Fever. 3. The Transmission of Q Fever by the Tick** *Haemaphysalis humerosa*.—*Australian Jl. Experim. Biol. & Med. Sci.* 1940. June. Vol 18. Pt. 2. pp. 103–118. With 2 figs. [Summary appears also in *Bulletin of Hygiene*.]

Haemaphysalis humerosa is known to be a natural vector of the virus of Q fever among animals, and the author has undertaken investigations to determine its importance in producing infection in man. He has studied the infection of the tick in larval, nymphal and adult stages, the transfer of virus from one stage to the next, and from adult to larva, the distribution of Rickettsia in the tick, and the infectiveness of the insect's tissues and faeces.

He found that laboratory-reared ticks would be infected by feeding on infected guineapigs in any of their stages, larva, nymph or adult. Next, that when larvae fed on similarly infected animals about half of them were found infected in later instars. Also, when nymphs were allowed to have an infective feed, 31 out of 56 were infected when they became adults. All the above were demonstrated by smears, by section of tissues and by inoculation into guineapigs.

As regards transfer of the virus from adults to the next generation : Sixteen female ticks which had been exposed to infection were examined for transovarial passage of the virus. In eight the virus was detected after oviposition was completed (not in the others which, perhaps, had not actually been infected). The progeny of one of these eight was found infected. A larger series of examinations is needed to confirm this. Within the infected tick, examination revealed no rickettsia in the salivary glands, nor in the intestine anterior to the

mid-gut and rare in the hind-gut. They seemed to multiply extracellularly within the lumen and in the free and degenerating cells of the hypertrophic epithelium. Inferentially, therefore, the faeces would probably be infective and this was found to be the case, and infection might occur even through unbroken skin.

The author summarizes his findings as follows :

" Larval, nymphal, and adult ticks were infected with rickettsiae by feeding them upon infected guinea-pigs during the febrile period. Slightly more than half of the ticks so treated were definitely proved to have acquired infection.

" Infection acquired by immature ticks was transferred through the succeeding metamorphosis or metamorphoses to the adult instar.

" Infected nymphal and adult ticks were found to infect about one-quarter of the host guinea-pigs upon which they were fed.

" Evidence suggestive of occasional transovarial passage of the virus was obtained, but not confirmed . . . . .

" In the tick host *R. burneti* appeared to be strictly confined to the intestinal epithelial cells and the lumen of the gut.

" The cell infection pattern was of the typhus type. The cytoplasm of infected cells was often packed with rickettsiae but the nuclei were never invaded.

" In partially engorged ticks multiplication of the rickettsiae appeared to take place both extracellularly and intracellularly.

" The faeces of infected ticks were found to be highly infectious, being capable of infecting guinea-pigs when applied to either abraded or unabraded skin. This method would appear to be the natural mode of infection of the vertebrate host.

" Triturated tissues of infected ticks were also found to infect guineapigs through unbroken skin. Some of the infections among abattoir workers may be attributed to such a mode of infection."

H. H. S.

Cox (Herald R.). *Rickettsia diaporica* **and American Q Fever.**—*Amer. Jl. Trop. Med.* 1940. July. Vol. 20. No. 4. pp. 463–469. [12 refs.]

A paper read at the Section of Rickettsial Diseases at the 3rd International Congress of Microbiology, New York, September, 1939.

The paper comprises a general review of the subject with reference to work already published. It is now suggested that the name American Q fever should be given to the disease in man since it is believed to be identical with Q fever in Australia. The parasite of the disease is a filterable Rickettsia-like body to which the name *R. diaporica* has been given by the American workers who first isolated it from ticks and who have shown that it is filterable. It has been found that this parasite is present in enormous numbers in the spleen and skin exudate of inoculated guineapigs and in the viscera and excreta of infected ticks and also in egg tissue cultures. From these it was possible to prepare practically pure emulsions of *R. diaporica*, and vaccines have been prepared which when inoculated into guineapigs have protected these animals against very large infective doses.

These emulsions have also been utilized for agglutination tests, and out of 72 blood samples sent in for examination from suspected cases 19 gave a positive reaction and also showed evidence of protective power. *R. diaporica* infection in nature has been demonstrated in ticks collected in Montana, Wyoming, Oregon and California. *D. H.*

HERTIG (Marshall). Cultivo de la *Bartonella bacilliformis* de un caso de verruga en el Ecuador. [**Cultivation of** *Bartonella bacilliformis.*]—*Bol. Oficina Sanitaria Panamericana.* 1940. Aug. Vol 19. No. 8. pp. 756–758. With 1 fig. English summary (9 lines).

"In the National Institute of Hygiene and Public Health of Lima four tubes containing semi-solid Noguchi's leptospira medium were inoculated with one specimen of blood from a suspected case of verruga peruana in Ecuador. On the fourth day colonies were observed which apparently corresponded to *Bartonella bacilliformis.* Subcultures showed on the fifth day typical groups suggestive of *Bartonella.* One South American monkey, probably of the *Cebus* species, was inoculated in the left eyebrow and left side of the abdomen with an Ecuadorian strain, and in the corresponding places on the right side with four Peruvian strains. Typical verruga peruana nodules developed on both sides."

MAYRAC (F.). Note sur une affection du type "fièvre des trois jours" à l'occasion d'une bouffée épidémique dans la circonscription de Hienghène (Nouvelle-Calédonie). [**Note on an Affection of the Type of "Three Day Fever" on the Occasion of a Sudden Outbreak in New Caledonia.**]—*Bull. Soc. Path. Exot.* 1940. Apr. 10. Vol. 33. No. 4. pp. 232–235.

The principal symptoms of the disease were severe headache and pains in the back and limbs with general weakness.

The European patients described their own feelings as "ça n'allait pas." Four distinct types of the disease were noted:—

1. Simple febrile type with initial rash and usually a recrudescence of fever on the 5th day.
2. Nervous type with intense headache.
3. Gastro-intestinal form.
4. A type in which the joint pains were the main feature.

In one case a confluent scarlatinal rash was the main symptom.

The infection appeared to have been introduced by imported labourers as the cases spread all over the colony from these people. It is suggested that the infection was carried by culicine mosquitoes.

D. H.

SHORTT (H. E.), PANDIT (C. G.), ANDERSON (W. M. E.) & RAO (R. Sanjiva). **Sandfly Fever Virus: Certain of its Properties and an Attempt at Prophylactic Vaccination.**—*Indian Jl. Med. Res.* 1940. Apr. Vol. 27. No. 4. pp. 847–863.

The authors have already shown that if the blood or serum from cases of sandfly fever is inoculated into monkeys a fever is produced in these animals, that mice inoculated with the cultured virus become infected, and that the virus can be passaged in the mice. This was done for seven generations but no increase of virulence was noted and mice inoculated from the passaged virus remained healthy and there was no evidence of pathological changes. Monkeys were also inoculated with large doses of cultured virus but without any apparent effects; it was found, however, that by culture of the virus on the chorio-allantoic membrane of eggs the presence of the virus in the blood of the monkeys could be demonstrated for an average of 11 days and for

19 days in one animal. Neutralizing power in the blood of these monkeys could also be demonstrated up to 69 days.

In actual cases of fever, and in convalescents, the presence of the virus in the blood can be demonstrated by egg culture up to three or four weeks after the onset of fever. The cultured virus was used as a vaccine from a 33rd passage in flask cultures. Eight volunteers received 2 doses of vaccine and eight others were used as controls. It was shown that virus was present in the blood of five of these inoculated persons and neutralizing bodies could also be demonstrated in the sera. Five of these men and the controls were then inoculated with the sera from cases of sandfly fever; the results were inconclusive, but some evidence was obtained to show that the presence of the virus in the blood coincided with a certain degree of immunity. *D. H.*

MALARIA.

Précis of Abstracts in this Section.

Hernan Mendez (p. 856) reports the first case of *P. ovale* infection to be found in Venezuela; the diagnosis was fully confirmed.

Sicé *et al.* (p. 857) show that *A. gambiae* sent to Marseilles in June were able to reproduce, and point out the danger of this species being accidentally transported to Southern France during the warm season. Barber (p. 857) shows that the invasion of new areas of Brazil by *A. gambiae* is followed quickly by epidemic malaria, largely because this mosquito prefers human blood, is very easily infected and can breed near human habitations. Control should be active in the long dry season when breeding places are relatively few. *P. falciparum* is the commonest parasite found.

Krishnan (p. 858) shows that *A. culicifacies* does not establish itself permanently or play a part in the transmission of malaria in Bengal. Although larvae are plentiful, adults are few in the spring, probably because the relative humidity is low. Later, the conditions become unfavourable on account of the rains, and *A. philippinensis* is probably responsible for malaria transmission, which increases from time to time with unusually heavy rainfall in the drier months. It is possible to predict an epidemic if heavy rainfall occurs in these months, and by the relative proportions of *P. falciparum* and *P. malariae* infections in February to May; in epidemic years the former predominates at that time. Conditions favourable to the breeding of *A. philippinensis* are noted.

Strickland *et al.* (p. 859) show that *A. maculatus*, though a vector in Malaya, cannot easily be infected in those parts of India in which it occurs, though Indian strains sent to Malaya are as easily infected there as are the local strains. The explanation appears to be the lower general infectivity of the Indian plasmodia. Toumanoff and Canet (p. 860) found that *A. hyrcanus* var. *sinensis*, usually not infected in Cochin-China, and markedly zoophilic in its habits, may be infected if no animals are available. Control measures should therefore include the securing of animal deviation of this and other secondary vectors.

Del Ponte (p. 860) records observations on *Anopheles pseudopunctipennis* in Jujuy (Argentine). It is by far the commonest anopheline present and is anthropophilic ; near a settlement there was an oöcyst rate of 1·7 per cent.

Crowell (p. 861) describes his method of rearing *A. quadrimaculatus* in an insectary.

Woino-Iassenetzky (p. 861) has studied the distribution of *P. falciparum* in the human body ; there is no uniformity in the distribution in the different organs. In fatal cases the accumulation of parasites is greatest in the vessels of the brain, and symptoms of coma only develop when the infected cells acquire the capability of accumulating in these vessels. Coma does not occur at the height of the paroxysms, but when the maturing ring forms disappear from the peripheral blood. Quinine has little influence on maturing schizonts.

In a study of multiple inoculations with *P. ovale*, Sinton (p. 862) shows that successive inoculations produce a higher degree of resistance to subsequent reinoculation than does a single infection. The antitoxic element of immunity develops more rapidly than the anti-parasitic element, though the latter is more durable.

Sicault and Messerlin (p. 862) consider the malaria of endemic countries to be a parasitic reticulo-endotheliosis. Three stages are recognized, a parasitaemic stage, a stage resembling splenic anaemia, and a final stage of malarial cachexia in which there is complete functional breakdown of the reticulo-endothelial tissues. The evolution is not regularly progressive and cure may be obtained in the first two stages. They (p. 862) describe the forms of reticulo-endotheliosis which they ascribe to malaria. Three forms are seen, one resembling splenic anaemia, one with Banti's syndrome and one of a pseudo-cirrhotic type. In all there is enlargement of the spleen and liver with anaemia, usually of the hyperchromic variety. Further details are given.

Fairley (p. 863) reports a case of a hitherto unrecorded haemolytic hypochromic anaemia associated with post-malarial splenomegaly of the Banti type. He suggests that the basis of the condition was escape of an intracellular lytic enzyme from a reticulo-endothelial system rendered pathological by chronic malaria. Crude liver extract and iron were successful in treatment.

Giordano (p. 863) reports a case of occlusion of the right branch of the anterior coronary artery, which he believes to have been due to malaria.

Boyd (p. 863) shows that infection with a large dose of sporozoites of *P. vivax*, compared with infection which follows a low dose, leads to a higher proportion of successful inoculations, a shorter incubation period, a longer and more severe clinical attack and greater liability to renewed activity after termination of the attack. In *P. vivax* malaria induced by blood inoculation Boyd (p. 864) found that renewed activity after the cessation of the primary attack has only been observed within a period of 8 weeks of such cessation. These infections thus differ markedly from naturally induced infections. The incubation period tends to be shorter, and the finding of parasites before the onset of symptoms more common, in these artificially induced infections.

Halawani and Sobky (p. 864) recommend the examination of thick drop preparations from haemolysed and centrifuged blood in malaria survey work.

GRIMES and LAVERGNE (p. 864) show that attacks of malaria are deleterious to patients with pulmonary tuberculosis in Madagascar, especially to patients with pneumothorax, in whom effusion is often produced. At a sanatorium in which reinfection with malaria is possible they advocate drug prophylaxis by means of rhodoquine taken for 5 days each month, and show that although the action of the drug is inhibitive rather than prophylactic, the desired results are obtained.

VARTAN and DISCOMBE (p. 865) describe a case of quinine poisoning. GARRA (p. 865) writes favourably of the treatment of malaria, both primary attacks and relapses, with antimony tartrate administered intravenously. The drug has a remarkable effect on the enlarged spleen.

CLUVER (p. 866) describes the standardized control of *A. gambiae* and *A. funestus* in Natal, where special attention is paid to the weekly spraying of houses with insecticide during the transmission season, with successful results. COVELL (p. 866) reports on the use of insecticidal sprays in villages in India and advocates their use where other measures are not feasible.

STRANGWAYS-DIXON (p. 867) has described the habits of *Gambusia affinis* and given practical hints on breeding. *C. W.*

KOBAYASI (Hidekazu), YOKOI (Keniti), KAWABE (Kunitaro) & TUTUMI (Senpo). **Parasitological Investigation in Hainan-Island. I. Epidemiological Studies on Malaria and Splenomegaly among the Natives of Hainan-Island, South-China.**—*Taiwan Igakkai Zassi* (*Jl. Med. Assoc. Formosa*). 1940. Mar. Vol. 39. No. 3. [In Japanese pp. 409–418. [20 refs.] English summary pp. 418–419.]

An examination of 1,031 persons in Hainan Island resulted in the discovery of 51 malaria parasite carriers; 118 had enlarged spleens. All three species of plasmodium were found, *P. vivax*, 20, *P. falciparum* 19, and *P. malariae* 12. *Norman White.*

HERNAN MENDEZ. *Plasmodium ovale.* Relación del primer caso observado en Venezuela. [**Report of a Case of** *Plasmodium ovale* **Infection in Venezuela.**]—*Publicaciones de la Division de Malariologia.* Ministerio de Sanidad y Asistencia Social. Caracas. 1939. Sept. 15. No. 3. 9 pp. With 5 figs. [15 refs.]

In this paper are described and figured the parasites found in a blood film taken from a child four years of age in Venezuela. The description and illustrations appear to justify the diagnosis of *P. ovale*. Professor Martin Mayer has confirmed the diagnosis. This is the first case of *P. ovale* infection reported from Venezuela. One case from Western South America was reported by MÜHLENS in 1934 [see this *Bulletin*, 1935, Vol. 32, p. 109]. *N. W.*

BATES (Marston) & HACKETT (L. W.). **The Distinguishing Characteristics of the Populations of** *Anopheles maculipennis* **found in Southern Europe.**—Reprinted from *Verhandlungen des VII. Internat. Kongresses f. Entom. 1938.* 1939. July. Vol. 3. pp. 1555–1569.

SICÉ (A.), SAUTET (J.) & ETHES (Y.). L'un des plus redoutables vecteurs du paludisme en Afrique, l'*Anopheles gambiae* Giles, 1902, est-il susceptible d'être transporté en France par les avions ?—[**Possibility of *A. gambiae* being introduced into France from Africa by Aeroplane.**]—*Rev. Méd. et Hyg. Trop.* 1939. July–Dec. Vol. 31. No. 4. pp. 137–139.

Adult female *A. gambiae* were sent by aeroplane from the French Sudan to Marseilles. They were sent in test tubes, without any elaborate precautions. A large proportion arrived alive. They laid eggs which developed normally in artificial breeding places, at ordinary temperature in Marseilles in June. The emerged females fed on blood, and were fertilized. These observations indicate that there may be a risk of this formidable vector of malaria being transported accidentally to Southern France during the warm season. *N. W.*

BARBER (M. A.). **The Present Status of *Anopheles gambiae* in Brazil.**—*Amer. Jl. Trop. Med.* 1940 Mar. Vol. 20. No. 2. pp. 249–267. With 1 chart & 1 map.

"There is no doubt that this invasion of gambiae threatens the Americas with a catastrophe in comparison with which ordinary pestilence, conflagration, or even war are but small and temporary calamities. Gambiae literally enters into the very veins of a country and may remain to plague it for centuries. Even the penetration of yellow fever into the Orient might be a lesser evil because its vector is domestic and more easily controlled." This quotation from an interesting report adequately describes the practical importance of its subject matter. A three months' study, at the close of the wet and well into the dry season, of *A. gambiae* on the frontier of its advance along the Jaguaribe River, above and below Iguatú in Ceará, yielded a wealth of valuable information. Malaria and malaria vectors were present in this part of Brazil before the invasion by *A. gambiae.* Malaria was transmitted by some species of *Nyssorhynchus*, probably *A. tarsimaculatus*, but parasite and spleen indexes were low. The advent of *A. gambiae* is followed, sometimes after the lapse of several weeks, by epidemic malaria. The preference of *A. gambiae* for human hosts, its susceptibility to malaria and its ability to breed near human habitations in rivers, rain-pools, ponds, and certain plantations are sufficient explanation. Almost all adults were found in inhabited dwellings. The relative prevalence of the three species of malaria parasite varied in different places : the proportion depends a good deal on chance gametocyte carriers. Among 231 positives in localities subject to *A. gambiae* transmission *P. falciparum* formed 56·7, *P. vivax* 38·1, *P. malariae* 1·3 and mixed *P. falciparum* and *P. vivax* infections 3·9 per cent. High infestations predominate and show no significant age incidence.

The prospect of preventing the spread of *A. gambiae* would be poor but for the long dry season, during which breeding places are limited : these must be sought for and freed from larvae. Adults must then be attacked. When profuse breeding is in progress human transportation should be limited as much as possible. Paris green, insecticidal sprays, mosquito netting, education and Gambusia can all help to limit the spread ; their use is discussed.

The present distribution of *A. gambiae* in Brazil has been fully investigated by R. C. SHANNON and his colleagues: their findings are to be published shortly. *N. W.*

KRISHNAN (K. V.). **Report(s) of the Professor of Malariology and Rural Hygiene.**—*Rep. All-India Inst. Hyg. Pub. Health. Calcutta.* 1937. pp. 23–28 and 1938 pp. 27–32. [Summarized in *Rev. Applied Entom.* Ser. B. 1940. Aug. Vol. 28. Pt. 8. pp. 153–154.]

"Investigations completed in 1937 showed that *Anopheles culicifacies*, Giles, does not establish itself permanently, or play a part in malaria transmission, in villages in Bengal, although the larvae are introduced every year in canal water. They appear during the non-malarious season in late winter and early spring, when the temperature of the water in the breeding places ranges between 80·6 and 84·2°F. and the ratio of dissolved carbon dioxide to oxygen is low (CO_2 being less than 0·2 and O_2 0·6 per 100,000 parts). These conditions are favourable for them. On the other hand, although the temperature (with maxima of 80–90 and minima of 70–80°F.) is favourable to the adults, the relative humidity is low (24–48 per cent.) and probably leads to the premature death of many of them. This suggestion is supported by the large numbers of larvae and small numbers of adults caught at this time. Later in the year, and particularly during the malaria season (July–October), when humidity as well as temperature is favourable for the adults, breeding conditions become unfavourable as a result of the rains. Not only does the temperature of the water rise to 90–95°F., but the ratio of CO_2 to O_2 is reversed (the former is increased to 1·0 per 100,000 parts and the latter reduced to less than 0·6). These conditions are, however, favourable for the breeding of *A. philippinensis*, Ludl., which is consequently responsible for malaria transmission.

"The abnormally high incidence of malaria at the field centre during 1937 is believed to have been due to the unusual amount of rain that fell during the usually dry month of February and led to the early breeding of *A. philippinensis*. Adults of this species began to appear in March instead of July. Breeding increased greatly with the onset of the monsoon rains, and adults were exceptionally prevalent between July and October. The sporozoite rate was remarkably high and, as a consequence, the malaria incidence also. The parasite index, the percentage of gametocyte carriers and the number of gametocytes per cubic millimetre all increased considerably and continued to do so over a longer period than usual. Reference to the history of malaria in the area revealed that such periodic increases occur once in 4–5 years, and a study of the rainfall records indicated that the increases were associated, as in 1937, with unusual rainfall during the drier months.

"During 1938, a further study of the causes of the periodic outbreaks of malaria that occur in the field malaria centre, which is situated in the Burdwan District, confirmed previous findings. In 1936, which was a non-epidemic year, the parasite rate was 5–10 per cent. during the dry season (March–June), 25–30 per cent. in the malaria season (July–October), and 10–15 per cent. during the cold weather (November–February). In 1937, the first of two years with unusual

rainfall in the dry months, no increase in malaria was noticed in the dry season, despite the fact that there was a marked increase in the Anopheline population and the conditions of temperature and humidity were favourable for transmission. This was probably attributable to the low numbers of gametocyte carriers. In the malaria season, however, both the number of Anophelines and the number of primary malaria cases increased. The parasite rate rose to 40–45 per cent. and this high rate persisted into the cold weather. The rainfall was again unusual during the dry months of 1938, and a severe epidemic occurred. Malaria was prevalent throughout the year and the parasite rate rose to 70 per cent. in the malaria season. It thus appears possible to predict an epidemic on the basis of the amount of rain falling in the dry months. It was also observed that more than 50 per cent. of the cases of malaria in February–May and July–September 1936 were due to *Plasmodium malariae*, whereas more than 50 per cent. in all months in the epidemic years were due to *P. falciparum*. The percentage due to *P. vivax* varied little during the three years. Thus, it appears possible, at least in this area, to forecast an epidemic rise in the ensuing malaria season from a study of the relative incidence of *P. malariae* and *P. falciparum* in the earlier months of the year (dry season).

"Larvae of *A. philippinensis* were found in only a minority of the reservoirs in the malaria centre, and a chemical analysis of the water in them indicated that a high ratio of CO_2 to O_2 was favourable to breeding and a low ratio unfavourable, the reverse of the findings recorded for *A. culicifacies* in the previous year. It was also observed that larvae of *A. philippinensis* did not occur in reservoirs in which *Euglena* or *Macrocystis* was present or in rice-fields."

STRICKLAND (C.), ROY (D. N.) & SEN GUPTA (S. C.). *Anopheles maculatus* **and Malaria.**—*Trans. Roy. Soc. Trop. Med. & Hyg.* 1940. Apr. 30. Vol. 33. No. 6. pp. 639–652. [21 refs.]

There has hitherto been some doubt as to the importance of *A. maculatus* as a vector of malaria in those parts of India where it prevails. Its ability to carry malaria in Assam, the Dooars and in the Terai has been claimed, on epidemiological grounds, by several observers. The authors of the present communication describe their attempts at infecting *A. maculatus* in the laboratory. In Calcutta, in the cold room, success was achieved. At laboratory temperature considerable difficulty was experienced; not one out of 18 could be infected in the laboratory though 13 out of 32 control *A. stephensi* became infected. In the Dooars attempts were more successful: during the epidemic season 15 out of 90 *A. maculatus* became infected. The infections were all with *P. falciparum*. All attempts at infecting the species with *P. vivax* failed. The success rate even in the Dooars was low, if the species play an important part in the spread of malaria, the incidence of which is very high. Batches of *A. maculatus* were sent to Malaya where their infectivity was compared with that of the local strain by KINGSBURY and his colleagues: the Malayan and Indian strains were found to be equally infective. The poor infection-rates of *A. maculatus* in India as compared with those obtaining in Malaya are ascribed to a lower general infectivity of Indian plasmodia.

N. W.

TOUMANOFF (C.) & CANET (J.). Quelques faits nouveaux au sujet de la transmission du paludisme dans la région des Terres-Rouges du Nord-Cochinchine. [**New Facts concerning the Transmission of Malaria in the Red-Soil Region of North Cochin-China.**]—*Bull. Soc. Path. Exot.* 1940. Mar. 13. Vol. 33. No. 3. pp. 188–194.

In the Red-Soil region of Cochin-China *A. minimus* and *A. jeyporiensis* are the chief vectors ; *A. hyrcanus* var. *sinensis* occurs but has not hitherto been found infected ; it is markedly zoophilic. In an isolated labour camp, devoid of domestic animals, *A. hyrcanus* var. *sinensis* has been found infected in the rainy season when *A. minimus* and *A. jeyporiensis* are but little prevalent. This observation indicates the desirability in certain rural centres of completing the specific measures taken to control *A. minimus* by securing the animal deviation of *A. sinensis* and other secondary vectors. This would be specially important if the artificial stagnation of water courses be employed to prevent *A. minimus* breeding. *N. W.*

DEL PONTE (E.). Observaciones sobre *Anopheles pseudopunctipennis* en La Mendieta, provincia de Jujuy. [**Observations on** *A. pseudopunctipennis* **at La Mendieta in the Province of Jujuy.**]—*Rev. Inst. Bact.* Buenos Aires. 1939. Vol. 9. No. 2. pp. 149–155. With 1 fig. [Summarized in *Rev. Applied Entom.* Ser. B. 1940. Aug. Vol. 28. Pt. 8. pp. 152–153.]

"Investigations were carried out in February–March 1939 on the importance of *Anopheles pseudopunctipennis*, Theo., as a vector of malaria at La Mendieta, a settlement in one of the most malarious districts in the province of Jujuy, Argentina, at an altitude of about 2,400 ft. Of 132 persons whose blood was examined, ten were infected with *Plasmodium vivax*, six with *P. falciparum*, and four with *P. malariae*, while one had a mixed infection. Of 45,818 Anophelines taken in 1938 in houses in the provinces of Tucuman, Catamarca, Salta and Jujuy, 45,569 were *A. pseudopunctipennis*, and in the author's investigations in La Mendieta this species was the only Anopheline taken in houses. It was the commonest species caught at nightfall near the settlement by means of human bait, the others being *A. albitarsis*, Arrib., *A. argyritarsis*, R.-D., and *A. tarsimaculatus*, Goeldi, and was the only one taken at night in the hills. It was also taken in a trap baited with a calf. It attacked man throughout the night in the hills, and the probability of contracting malaria depends chiefly on the percentage of infection in it. No infection was observed in 56 females of *A. pseudopunctipennis* taken in the hills with human bait, but of 644 taken near the settlement 1·7 per cent. showed oöcysts on the stomach. When batches of not less than 10 females of *A. pseudopunctipennis* were allowed to feed on carriers of gametocytes of *P. vivax*, and dissected 10 days later, no infection was observed. The morphology of the adults of both sexes, the larvae and the eggs of *A. pseudopunctipennis* is discussed, and a key is given to the pupae of the four Anophelines taken. Other mosquitos caught in the hills at La Mendieta included *Aëdes scapularis*, Rond., *Haemagogus* sp., and *Psorophora ferox*, Humboldt, which are potential vectors of jungle yellow fever. Larvae and adults of *C. fatigans*, Wied. (*quinquefasciatus*, auct.) and *Aëdes aegypti*, L., were taken in houses."

CROWELL (Robert L.). **Insectary Rearing of** *Anopheles quadrimaculatus.* **(A Preliminary Report.)**—*Amer. Jl. Hyg.* 1940. July. Vol. 32. No. 1. Sect. C. pp. 12–20. With 3 figs.

The author describes methods of rearing and maintaining a large laboratory stock of *Anopheles quadrimaculatus* : it has been in captivity since 1932, and had gone through nearly 90 generations at the time of writing. He has simplified a technique previously described by BOYD, so that one man can now do all the necessary work in two hours daily, producing about 1,500 larvae a week for tests of larvicides, and maintaining a population of three to five thousand adults.

The method (fully described and illustrated) is based on feeding larvae with pulverized dog biscuit, and rearing adults in a cage of about one metre cube ; they are fed on a clipped rabbit.

P. A. Buxton.

WOINO-IASSENETZKY (M. V.). La distribution des parasites dans l'organisme de l'homme au cours de la malaria tropicale. [**Distribution of Parasites in the Human Body in Tropical Malaria.**] —*Med. Parasit. & Parasitic Dis.* Moscow. 1939. Vol. 8. No. 6. [In Russian pp. 3–28. With 7 figs. [25 refs.] French summary pp. 28–29.]

The author's studies are based on 139 fatal cases of acute *P. falciparum* malaria. In thirty of these clinical details were sufficient to permit of a comparative study of the distribution of the parasites in the body in relation to the symptoms displayed. The distribution of parasites is very varied. In fatal cases the greatest accumulation of parasites occurs in the vessels of the brain, and in the adipose cellular tissue, of the mucosa of the alimentary canal and of the pancreas ; in pregnant women there is also an accumulation in the inter-villous spaces of the placenta and in the capillaries of the breast. In cases in which coma has not occurred there is no accumulation of mature schizonts in the blood-vessels of the brain ; it would appear that in such cases the parasites mature in the vessels of the intestinal mucosa, of the adipose cellular tissue and of other tissues. Symptoms of coma only develop when infected red-cells acquire, under certain influences, the capacity of accumulating in the cerebral capillaries. Involvement of the nervous system is not dependent on the numbers of parasites present in the body ; when the number is very low, however, coma is unlikely to occur. Cerebral cases in which but few parasites are found at autopsy are explicable by the action of quinine. There is no uniformity of distribution of parasites in the different organs ; large schizonts are found at the site of large parasite accumulations ; elsewhere small unpigmented forms predominate as in the peripheral blood. Coma does not occur at the beginning or at the height of a paroxysm but only after the lapse of 20 to 24 hours after the appearance of the generation of parasites that is responsible for the condition and when the maturing ring forms disappear from the peripheral blood. On maturing schizonts quinine appears to have but little influence. Gametocytes are formed exclusively in the bone-marrow and the spleen ; they are very rarely seen at the sites of large parasite accumulations.

N. W.

SINTON (J. A.). **Studies of Infections with *Plasmodium ovale*. V. The Effects of Multiple Inoculations upon the Degree and Nature of the Immunity developed.**—*Trans. Roy. Soc. Trop. Med. & Hyg.* 1940. Apr. 30. Vol. 33. No. 6. pp. 585–595. [24 refs.]

This is a study of the reactions of the human host, as shown by parasitic and febrile response, to successive inoculations of *P. ovale*. Ninety-eight cases, in which the primary attack was not modified by early treatment, are considered; of these 40 subsequently received a second inoculation, 12 of these a third, and one a fourth inoculation. It was shown that successive inoculations produce a progressively higher degree of resistance to subsequent reinoculation than does a single infection. In this respect *P. ovale* resembles the other species of human plasmodia. The anti-toxic element of the immunity, as evidenced by diminished febrile response to infection, appears to be developed more rapidly than the anti-parasitic element, and more rapidly than in the case of infection with the other three human plasmodia. In the absence of renewed infection the anti-parasitic element of the immunity appears to be more durable than the anti-toxic element. *N. W.*

SICAULT (G.) & MESSERLIN (A.). L'évolution de la réticulo-endothéliose palustre. (1er mémoire.) [**Evolution of Malarial Reticulo-Endotheliosis.**]—*Bull. Soc. Path. Exot.* 1940. Apr. 10. Vol. 33. No. 4. pp. 272–279.

In this paper malaria of endemic countries is considered as a parasitic reticulo-endotheliosis. Three stages are recognizable in its evolution. The first stage corresponds to the parasitaemic period of the disease and the reticulo-endothelial changes are in large part secondary to the blood infection. In the second stage there is loss of function of the reticulo-endothelial tissue; the clinical picture is that of splenic anaemia. In the third stage there is a complete functional break-down of reticulo-endothelial tissues and of parenchyma functionally associated with them; the clinical picture is that of malarial cachexia. This evolution is not regularly progressive; successive exacerbations may be separated by long intervals, and spontaneous or therapeutic cure may occur during the first two stages of the process. *N. W.*

SICAULT (G.) & MESSERLIN (A.). Quelques formes souvent meconnues de la réticulo-endothéliose palustre. (2e mémoire.) [**Some Frequently Unrecognized Forms of Malarial Reticulo-Endotheliosis.**]—*Bull. Soc. Path. Exot.* 1940. Apr. 10. Vol. 33. No. 4. pp. 280–287.

The observations in this paper are based on the study of 82 patients' mostly middle aged, all resident in hyperendemic malarial regions of North Africa. They comprise three clinical types: (1) a splenic anaemia type with splenomegaly and an intense anaemia, the red-cell count often being below 1,500,000; (2) a type exhibiting Banti's syndrome with splenomegaly, moderate anaemia and terminal ascites, and (3) a pseudo-cirrhotic type with enlarged spleen and liver and occasional attacks of ascites. In all these cases the authors believe that the pathological conditions were directly, or indirectly, attributable to malaria. Features common to all cases were splenomegaly, well

below the umbilicus ; anaemia generally of the hyperchromic variety ; enlargement of the liver with disturbance of its functions and the frequent occurrence of ascites ; general weakness, lowered arterial tension and occasional febrile attacks ; and a humoral syndrome consisting of increase of serum euglobulin precipitable by distilled water, a decrease of blood cholestrin, and a frequent increase in the bilirubin content of the blood.

Four illustrative cases are described. *N. W.*

FAIRLEY (N. Hamilton). **A Peculiar Haemolytic Hypochromic Anaemia associated with Post-Malarial Splenomegaly of Banti's Type.**—*Trans. Roy. Soc. Trop. Med. & Hyg.* 1940. Aug. 16. Vol. 34. No. 2. pp. 173–186. With 2 graphs.

A detailed account of a case of a hitherto unrecorded haemolytic hypochromic anaemia associated with post-malarial splenomegaly of Banti's type. Injections of refined liver extract with iron therapy and blood transfusions failed, but crude liver extracts and iron *per os* unexpectedly produced satisfactory haemopoiesis. Splenectomy was followed by a haemolytic megalocytic erythroblastic anaemia. It is suggested that the basis of the condition was escape into the blood of an intracellular lytic enzyme from pathological reticulo-endothelium following chronic malaria. *W. P. Kennedy.*

GIORDANO (Francesco). Un caso di "blocco di branca destra" in malarico cronico. [**Coronary Occlusion in Chronic Malaria.**]—*Riforma Med.* 1940. Mar. 30. Vol. 56. No. 13. pp. 408–410. With 2 figs.

The author directs attention to two previous publications in which he described the frequency of cardiac involvement in malaria infections, characterized for the most part by cardiovascular dilatation with disturbance of rhythm. He describes in detail the case of an old chronic malaria patient in whom the cardiac symptoms are attributed to occlusion of the right branch of the anterior coronary artery. The report is illustrated with electro-cardiograms and radioscopic appearances. He believes malaria to have been the primary cause of the condition. *N. W.*

BOYD (Mark F.). **The Influence of Sporozoite Dosage in Vivax Malaria.**—*Amer. Jl. Trop. Med.* 1940. Mar. Vol. 20. No. 2. pp. 279–286.

Anopheline mosquitoes employed in inoculation are dissected subsequent to their application and only those containing sporozoites in the salivary glands are recorded as having effected the inoculation. The quantitative infection of various lots of mosquitoes is determined by the number of oöcysts found in the stomachs of individuals of the lots that have been dissected during the maturation of infection. In this paper a comparison is made of cases infected with lots of mosquitoes of which one at least harboured more than 100 cysts per stomach, with cases infected with lots of mosquitoes of which none were observed with more than 50 cysts. The author's conclusions are as follows :—

"The dosage of sporozoites of *P. vivax* with which a susceptible person is inoculated, as deduced from the quantitative infection of a lot of mosquitoes and the number of specimens of such a lot employed for an

inoculation, exerts a significant effect on the subsequent infection. If the dosage is small, the proportion of unsuccessful inoculations will be high. The duration of the incubation period tends to vary inversely with the dose of sporozoites received. The duration of the clinical attack appears to vary directly with the dose of sporozoites, and inversely with the length of the incubation period.

"Renewed activity is most frequent following induced termination of attacks produced by heavily infected mosquitoes. There is some tendency for patients inoculated with such mosquitoes to experience a greater number of severe paroxysms than do those inoculated with lightly infected mosquitoes." *N. W.*

BOYD (Mark F.). **Some Characteristics of Artificially Induced Vivax Malaria.**—*Amer. Jl. Trop. Med.* 1940. Mar. Vol. 20. No. 2. pp. 269–278.

The observations reported are based on the records of 54 patients successfully inoculated with trophozoites of *P. vivax*. Intravenous inoculation of not more than 10 cc. of the donor's whole blood, citrated, was the method employed. With such small volumes of blood no danger arises from the possible incompatibility of donor and recipient. The incubation period tends to be shorter than that following natural infection. The detection of parasites before the onset of clinical symptoms is more common than in the case of natural inoculation. The mean duration of the primary attack in susceptible persons differs but little from that of naturally inoculated persons, nor do the characteristics of the paroxysms differ. Renewed activity after the cessation of the primary attack has only been observed within a period of 8 weeks from such cessation: in this respect artificially induced cases differ markedly from naturally induced infections. *N. W.*

HALAWANI (A.) & SOBKY (M. F.). **On the Efficacy of the Method of Concentration of Blood Parasites in the Diagnosis of Malaria.**—*Jl. Egyptian Med. Assoc.* 1939. Sept. Vol. 22. No. 9. pp. 509–516.

To concentrate blood parasites the authors take 0·5 cc. of blood in 10 cc. of 1 per cent. acetic acid (or formalin) solution. The solution is centrifuged for 10 minutes at 3,500 revolutions per minute The supernatant fluid is pipetted off and a drop of the sediment examined. One hundred cases suspected of malaria, were examined. The examination of two thick drop preparations from each case showed 52 infected: subsequent examination by the concentration method revealed parasites in 80. Only 11 crescent carriers were found by the thick drop method, 31 by the concentration method. The authors recommend the method for malaria survey work. *N. W.*

GRIMES (Ch.) & LAVERGNE (J.). Action prophylactique antipalustre des médicaments gamétocides employés seuls, en particulier chez les bacillaires avancés. [**Antimalarial Prophylactic Action of Gametocide Drugs, with Special Reference to Advanced Cases of Pulmonary Tuberculosis.**]—*Bull. Soc. Path. Exot.* 1940. Mar. 13. Vol. 33. No. 3. pp. 194–201.

This paper is based, for the most part, on experience in a tuberculosis sanatorium in Madagascar, in which nearly all patients have suffered from malaria, and in which reinfection with malaria is possible An

attack of malaria is a serious complication of tuberculosis and may undo much of the good conferred by sanatorium treatment. In pneumothorax cases the complication is specially grave; after a febrile malaria attack a large pulmonary effusion is very likely to occur. The clinical prophylaxis of malaria thus assumes considerable importance in a tuberculosis sanatorium.

The authors have used rhodoquine, praequine and rhodopraequine, drugs which have gametocidal and antisporogonic properties. Of these rhodoquine is the best tolerated by enfeebled patients, and is the most effective. They recommend the administration of a five-day course of rhodoquine, 0·03 gm. a day, every month. During 1938, a year in which these drugs were systematically given, none of the 39 pneumothorax cases had a large pleural effusion, a complication which was of frequent occurrence in previous years. The authors realize that the dysgonic action of these drugs is inhibitive rather than prophylactic, but they are effective in securing the desired result. Quinacrine is ill tolerated by this class of patient, and prophylactic quinine is of small value. *N. W.*

VARTAN (C. K.) & DISCOMBE (G.). **Death from Quinine Poisoning.**—*Brit. Med. Jl.* 1940. Mar. 30. pp. 525–526. With 2 figs. on 1 special plate.

A woman, aged 34, who had always lived in or near London and who had never had malaria took 16 quinine pills as an abortifacient. The amount of quinine sulphate ingested approximated 6·08 gm. She developed symptoms very closely resembling those of severe blackwater fever and died after nine days' illness. The difficulties of diagnosis and the necropsy findings are fully described. *N. W.*

GARRA (Giuseppe). Cura della malaria con il tartaro stibiato secondo il metodo de Nunno. (Studio clinico sui nazionali e sugli indigeni.) [**Treatment of Malaria with Antimony Tartrate according to de Nunno's Method.**]—*Riforma Med.* 1940. Mar. 30. Vol. 56. No. 13. pp. 410, 413–18. With 2 figs.

A record of the results of treatment of 29 cases of malaria by intravenous injections of antimony tartrate according to the method devised by DE NUNNO [see this *Bulletin*, 1939, Vol. 36, p. 500]. The observations were made in Italian East Africa; twelve patients were Italian, seventeen natives. There were 8 *P. vivax* and 13 *P. falciparum* infections; in 8 cases no parasites were found. The author thinks highly of the method of treatment, which was well tolerated. It has a remarkable influence on the enlarged spleen. The action of the drug appears to be more prompt on primary infections than on relapses, but the majority of cases treated were those in which the more usual antimalaria remedies had failed to effect a cure. The effect of the treatment on the patients' general condition was most beneficial.

N. W.

BULLETIN DE L'OFFICE INTERNATIONAL D'HYGIÈNE PUBLIQUE. 1940. May–June. Vol. 32. No. 5–6. pp. 572–582.—Le paludisme dans la marine marchande. [**Malaria in the Mercantile Marine.**]

This is a French translation of a pamphlet prepared for the guidance of captains of British ships not carrying medical officers. It is a

supplement to the Ship's Captain's Medical Guide. Measures that should be taken to protect the ship's company from mosquito bites in infected ports, quinine prophylaxis, the symptoms of malaria, and the treatment of an acute attack are simply and clearly described.

N. W.

HOWARD (S. C.). **The Practical Application of Anti-Malarial Measures on Malayan Estates.**—*Bull. Inst. Med. Res. Federated Malay States.* 1939. No. 2. pp. 1–12. With figs. 1–6 on 3 plates.

HOLMES (W. E.). **The Control of Urban Malaria (Kuala Lumpur).**—*Ibid.* pp. 13–17. With figs. 7–9 (2 on 1 plate & 1 folding).

WILSON (T.). **The Control of Rural Malaria in Malaya.**—*Ibid.* pp. 18–22. With figs. 10–11 on 1 plate.

These are three lectures given in an international course of malariology arranged by the League of Nations Health Organisation in Singapore. Those conversant with reports published in recent years concerning much admirable work on malaria control and prevention carried out in Malaya will find nothing new, but the lectures are useful and interesting summaries of successful work in this field. *N. W.*

CLUVER (F. W. P.). **Malaria Control in Natal and Zululand.**—*South African Med. Jl.* 1940. Mar. 23. Vol. 14. No. 6. pp. 113–117. Also in *Public Health.* Johannesburg. 1940. Aug. Vol. 4. No. 11. pp. 16–19, 23, 25.

Malaria has always been very prevalent in the north coastal portion of Natal, but prior to the epidemic of 1932 malaria control was not taken very seriously. The two vectors of malaria are *A. gambiae* and *A. funestus*, both almost exclusively anthropophilic and house-frequenting; their breeding habits are, however, very dissimilar. Since 1933 control methods have been standardized. Great prominence is given to the destruction of adult mosquitoes in dwellings by means of an insecticidal spray. Houses are sprayed weekly throughout the transmission season with a mixture of pyrethrum and commercial paraffin; sometimes more frequently. Very great success has attended the application of these measures. Morbidity and mortality statistics are not available to show the diminution of the incidence of malaria, but diminished hospital admissions, fewer positive blood films submitted for examination, and above all, a marked improvement in labour and economic conditions are sufficient testimony to the success achieved. *N. W.*

COVELL (G.). Désinsectisation des habitations au moyen de pulvérisation de liquide insecticide à base de pyrèthre comme mesure de prophylaxie antipaludique. [**Malaria Control with Insecticidal Sprays.**]—*Bull. Office Internat. d'Hyg. Publique.* 1940. May–June. Vol. 32. No. 5–6. pp. 564–569.

This report describes an attempt at controlling malaria in two villages close to Delhi, Nizamuddin and Jangpura, with an insecticidal spray, a 1 in 20 dilution of Pyrocide 20 in kerosene oil. The first year's observations have already been published [see this *Bulletin*, 1938, Vol. 35, p. 901]. Although it is not possible to determine with accuracy the value of the operations in these villages the author

considers that with adequate organization and supervision the method merits further trial, especially in small isolated communities where other measures are not economically feasible. The cost of spraying compares favourably with that of temporary antilarval measures.

N. W.

STRANGWAYS-DIXON (D.). *Gambusia affinis holbrookii*: **Imported Antimalarial Fish in East Africa.**—*East African Med. Jl.* 1940. Mar. Vol. 16. No. 12. pp. 450–455.

A description is given of *Gambusia affinis holbrookii*, and of its life history and habits. There were difficulties experienced in acclimatizing these fish at an altitude of 6,500 feet in Kenya but the difficulties have been surmounted and there are now distribution centres in most provinces in Kenya. The paper contains practical hints for the breeding and the distribution of the fish. *N. W.*

BACILLARY DYSENTERY AND INTESTINAL PROTOZOAL INFECTIONS.

PRÉCIS OF ABSTRACTS IN THIS SECTION.

Bacillary Dysentery.—BURKITT (p. 867) uses Pulv. rhei in the treatment of bacillary dysentery, giving one teaspoonful with water every hour until the rhubarb appears in the stool, and then less frequently. He reports that with this treatment none of his patients died in a period of 21 years during which he employed it in Nairobi. GORLITZER (p. 868) reports success in the treatment of Shiga-Kruse infections by means of prontosil. Improvement is obtained even in cases of long standing. DRUMMOND (p. 868) has obtained success in treatment with soluseptasine and advises further trials of this drug.

Protozoal infections.—SPADARO (p. 869) reports that human giardiasis is common in Eritrea, and thinks that in some way it is related to infection in rats.

GRÜNEIS and PILGERSTORFER (p. 869) have studied the anaemia associated with infections with *G. intestinalis* and conclude that it is not directly the result of infection but that it is caused by long standing inflammation of the intestine, particularly of the duodenum. Eradication of the flagellates without iron therapy did not cure the anaemia, but the administration of iron even in the continued presence of the flagellates resulted in improvement. It seems, therefore, that though the flagellates are not responsible for the inflammation, the latter is favourable to them.

LAVIER (p. 869) has described the method of binary fission of *Giardia muris*.

GALIZZI (p. 870) reports cure, by means of atebrin or other acridine derivatives, of 5 cases of infection, but definite cure can only be accepted after long observation.

Experimenting on himself with *Chilomastix mesnili*, WESTPHAL (p. 870) concludes that the intensity of infection is directly proportionate to the degree of failure of carbohydrate digestion in the intestine, and that the presence of the flagellate is the result rather than the cause of any intestinal derangement with which it is

associated. DE MURO and CURATOLO (p. 871), however, state that *Chilomastix mesnili* produces a catarrhal colitis of a putrefactive type.

C. W.

BURKITT (R. W.). **Treatment of Acute Bacillary Dysentery.** [Correspondence.]—*Brit. Med. Jl.* 1940. May 18. p. 831. [Summary appears also in *Bulletin of Hygiene.*]

As the outcome of a discussion on bacillary dysentery held at the Royal Society of Tropical Medicine & Hygiene in March, Dr. Burkitt has communicated a letter to the *British Medical Journal* in which he advocates the use of Pulvis rhei in cases of infection by bacillary dysentery, regardless of the infecting bacterium. He gives a teaspoonful (not a measured drachm) with a draught of water every hour till rhubarb appears in the stool, then less frequently for a few days. He suggests that it acts by its cholagogue effect and, possibly, as a stimulant of intestinal secretions. The acute symptoms become modified as soon as the drug makes the stool red ; the abdominal tenderness and rigidity are less, pain disappears, the temperature, pulse and facies improve. The author does not say how many cases were under his care but states : " In Nairobi from 1911 to 1917 I never saved a case in any race. From 1917, when I began to use rhubarb, to a couple of years ago [*i.e.* 1938, or a period of 21 years] when I left East Africa, I never lost a case in either adult or child." Also in a hospital of 1,500 beds dysentery deaths fell from 30 daily to three when this form of treatment was adopted.

H. H. S.

GORLITZER (V.). Ueber Behandlung von Bazillen-Dysenterie mit Prontosil. [**Treatment of Bacillary Dysentery by Prontosil.**]—*Schweiz. Med. Woch.* 1940. Mar. 30. Vol. 70. No. 13. p. 281.

The author employed prontosil in the following manner for treating cases of bacillary dysentery : one tablet [dose not stated, ? 0·5 gm.] thrice daily for three days ; on the evening of the third day 10 drops of Tinct. opii ; fourth to seventh days one tablet of prontosil twice daily and, thrice daily, a charcoal tablet. Whether prontosil rubrum or album was used seemed to make no difference, the results were equally good.

All the patients cited had Shiga-Kruse bacteria in the stools. Early cases reacted " in a few days " ; fever dropped, stools became normal, and discomfort ended. Even those who had been ill for a year and had received various forms of treatment, including vaccines, within a fortnight were free of fever and pain. One patient with long-standing disease and a fistula between the bowel and the bladder, fever, emaciation, abdominal pain, and blood-stained stools, lost the fever in a week and the pain disappeared, the stools became formed and appetite improved and in a fortnight the improvement and gain in strength were such that the patient was able to get up. The author prescribes a soft, bland diet for six weeks, after which time examination failed to reveal dysentery bacteria.

H. H. S.

DRUMMOND (J.). **Sulphonamides in Treatment of Bacillary Dysentery.** [Correspondence.]—*Brit. Med. Jl.* 1940. Sept. 14. pp. 367–368.

The author has treated 7 cases of bacillary dysentery (3 Flexner, 2 Morgan, 1 *asiaticus* and 1 pseudo *carolinus*) with 5 cc. soluseptasine

parenterally each day combined with 6 tabloids orally for 5 days. The symptoms were typical, but in all the condition of the bowel became normal within 10 days. The series is small but the results have been so impressive that the author advocates further trials of the method. *C. W.*

SPADARO (Orazio). Osservazioni sulla lambliasi in clima tropicale. [**Lamblia Infections in the Tropics.**]—*Rass. Sanitaria dell' A.O.I.* Addis Ababa. 1939. Sept. 9. Vol. 1. No. 3. pp. 60–64. With 1 chart. [38 refs.]

The author points out that lamblia infections are common in Massaua, Eritrea, particularly during the winter months. He seems to think that the human infections are in some way related to those of rats, though it has been established that *Giardia muris* is distinct from the human parasite. *C. M. Wenyon.*

GRÜNEIS (Paul) & PILGERSTORFER (Erich). Ueber das Zustandekommen anämischer Zustände bei der Lamblieninfektion des Menschen. [**Causation of Anaemic Conditions in Human Lamblia Infections.**]—*Wien. Klin. Woch.* 1939. Nov. 3. Vol. 52. No. 44. pp. 991–993. [39 refs.]

The authors refer to the statement so often made that lamblia infections are liable to be followed by anaemia. From this point of view a number of cases harbouring the flagellate were carefully investigated, a study of the blood and bone marrow changes being made. It is concluded that the flagellate alone is unable to bring about an anaemia but that it is the occurrence of long standing inflammatory conditions of the intestine, particularly of the duodenum, which is directly responsible. The type of anaemia corresponds with this view, as also does the improvement following the administration of iron even in the continued presence of the flagellates. In one case there was added a cirrhotic condition of the liver which was reflected in the type of anaemia present. The eradication of the lamblia infection by atebrin or a new acridine derivative which was tried was not followed by disappearance of the anaemia in the absence of iron therapy. It seems, therefore, that the flagellate itself is not responsible for the inflammatory condition of the intestine, which, however, is favourable to its maintenance as well as to the development of the anaemia. If the condition of the intestine is improved by modern methods of treatment the response to iron therapy is more marked. *C. M. W.*

LAVIER (G.). Le mode de division des formes végétatives dans le genre *Giardia*. [**The Method of Division of** *Giardia*.]—*C. R. Soc. Biol.* 1939. Vol. 132. No. 25. pp. 452–455. With 7 figs.

In this paper the author describes with the aid of illustrations the complicated process of binary fission of *Giardia muris*. This is the first time that all stages of this division have been depicted, though certain of them had previously been given for the human parasite by WENYON and O'CONNOR. *C. M. W.*

GALIZZI (João). Novo tratamento da giardiase humana. [**New Treatment of Human Lambliasis.**]—*Brasil-Medico.* 1939. Mar. 4. Vol. 53. No. 10. pp. 329–334. [12 refs.] English summary.

The paper reviews the clinical symptoms associated with giardia infections and gives an account of five cases which were freed of the flagellates by atebrin or other acridine derivatives. The author reports only the parasitological cure for, as he says, other digestive complaints were treated at the same time. He considers that a definite cure can be accepted only after a long and continuous observation. *C. M. W.*

VÉGHELYI (Peter V.). **Giardiasis.**—*Amer. Jl. Dis. Children.* 1940. Apr. Vol. 59. No. 4. pp. 793–804. With 1 chart. [17 refs.]

This paper appears to be an amplification of the one already reviewed in this *Bulletin*, 1940, Vol. 37, p. 376. *C. M. W.*

WESTPHAL (Albert). Beziehungen zwischen Infektionsstärke und "Krankheitsbild" bei Infektionen mit *Chilomastix mesnili* und anderen Dickdarmflagellaten. [**The Relationship between Intensity of Infection and the Clinical Symptoms in Infections with *Chilomastix mesnili* and Other Intestinal Flagellates.**]—*Ztschr. f. Hyg. u. Infektionskr.* 1939. Oct. 21. Vol. 122. No. 2. pp. 146–158. With 2 figs. [14 refs.]

By a series of ingenious experiments carried out upon himself the author has attempted to obtain evidence of the pathogenicity of the intestinal flagellate *Chilomastix mesnili*. In the first place he infected himself by the ingestion of encysted forms of the flagellate which he harboured for a considerable time without any symptoms. The ingestion of pure reduced iron powder gave rise to diarrhoea associated with a great increase in the number of flagellates. The acquired infection disappeared, whereupon the author again ingested cysts from another case. On this occasion again infection took place but was associated with a diarrhoeic condition. It might have been thought that this was proof of the pathogenicity of the flagellate, but from the stool was isolated a strain of *Bact. coli* which proved to be pathogenic to cats. After the subsidence of the diarrhoea the flagellate infection persisted. It was noted that the degree of infection varied from time to time but was most intense when the stool was soft. This was true also of an accompanying *Enteromonas hominis* infection. As there were no further intestinal symptoms it appeared that the fluctuations in the infection were due to changes in the intestinal contents. In another experiment the diet was arranged so that a high proportion of ingested carbohydrate arrived undigested in the large intestine. This again was associated with a marked increase in the intensity of the infection. It would seem that the intensity of the infection is directly proportional to the degree of failure of carbohydrate digestion in the intestine and that the presence of the flagellate is the result rather than the cause of any intestinal derangement with which it is associated. *C. M. W.*

DE MURO (P.) & CURATOLO (A.). Colite catarrale da *Chilomastix mesnili*. [**Catarrhal Colitis due to** *C. mesnili*.]—*Policlinico*. Sez. Prat. 1940. Feb. 12. Vol. 47. No. 6. pp. 213–16, 219–20. [38 refs.]

The literature of *Chilomastix mesnili* infection is reviewed and the symptoms of nine patients harbouring this parasite are discussed. It is concluded that the flagellate is the cause of a catarrhal colitis of a putrefactive type. *C. M. W.*

TROPICAL OPHTHALMOLOGY.

A REVIEW OF RECENT ARTICLES. XXXVI.*

Eyelids.—Mycetoma of the lid is reported by ALDRIDGE and KIRK[1] in a Nubian girl, from the district of Halfa. The granuloma formed a painless tumour the size of a marble in the upper lid ; the skin was not involved. The histopathological appearance was typical of mycetoma.

Conjunctiva.—Cases of *streptothrix infection of the conjunctiva* have been described by BADIR[2], FADEL[3] and EL BAKLY[4]. The disease is characterized by the formation of yellowish, vascularized, firm nodules in the bulbar conjunctiva, which involve the episcleral tissue. Cure is effected by the excision of some or of all the nodules. Infection seems to occur from grain dust when the harvest is being gathered.

Trachoma.—TABORISKY[5] speaking from experience of the disease in Russia, Germany, Austria and Palestine found the clinical picture in all countries identical. Follicular development and papillary hypertrophy are very marked in the trachoma of North Russia ; this may be due to an excessive lymphatism of the inhabitants caused by residence in comparatively sunless cities. In the Ukraine the people of rural districts suffered more severely than urban populations. SHIGA[6] has given some account of the disease in Japan. The index among elementary school-children has fallen from 17 per cent. in 1913 to 12 per cent. in 1933, girls being slightly more affected than boys. Amongst army recruits the incidence decreased from 23 per cent. in 1909 to 9·67 in 1933. Here, as elsewhere, the disease is most prevalent amongst the poverty-stricken. In districts where trachoma prevails follicular conjunctivitis is rare, but trachoma is rare in some districts where folliculosis is common. A Trachoma Prevention Law exists

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* For the 35th of this series see Vol. 37, pp. 447–450.

[1] ALDRIDGE (J. S.) & KIRK (R.). Mycetoma of the Eyelid.—*Brit. Jl. Ophthalm.* 1940. May. Vol. 24. No. 5. pp. 211–213. With 1 fig.

[2] BADIR (G.). Streptothrix Infection of the Conjunctiva.—*Bull. Ophthalm. Soc. Egypt.* 1939. Vol. 32. pp. 99–103.

[3] FADEL (A. E. L.). A Case of Streptothrix of the Conjunctiva.—*Bull. Ophthalm. Soc. Egypt.* 1939. Vol. 32. pp. 104–105.

[4] EL BAKLY (M. A.). Streptothrix of the Conjunctiva.—*Bull. Ophthalm. Soc. Egypt.* 1939. Vol. 32. pp. 106–113.

[5] TABORISKY (J.). Trachoma in Northern, Southern Russia and in Palestine.—*Rev. Internat. du Trachome.* 1939. Oct. Vol. 16. No. 4. pp. 201–208.

[6] SHIGA (H.). Trachoma in Japan with Special Reference to Public Health.—*Amer. Jl. Ophthalm.* 1940. Mar. Vol. 23. No. 3. pp. 306–314. [10 refs.]

in the country; this provides for the care of patients and for the prevention of the spread of contagion.

Experiments conducted by JULIANELLE and SMITH[7] failed to furnish any evidence of the rickettsial origin of trachoma or to confirm the observations of some other workers regarding the occurrence of a positive Weil-Felix reaction in the disease. JULIANELLE[8] has also investigated the occurrence of inclusion bodies and has found them in thirty-three per cent. of 602 cases of trachoma of all types. Fifty per cent. of those in whom the duration of the disease was six months were positive, but at ten years the rate fell to zero. Tissues containing inclusions were twice as infective as those without. The infectivity experiments were carried out on monkeys. REIS[9] who has reported on trachoma in Poland, also concluded that the Weil-Felix reaction can give no help in the diagnosis of the disease, since her experience shows that an equally high percentage of positive reactions may be found in non-trachomatous persons suffering from gonorrhoea and in perfectly healthy people.

Many reports have been made on the treatment of trachoma by the use of sulphonamide compounds; some of these are enthusiastic and others less so. The view expressed by MACCALLAN[10] seems suitably to sum up the situation. He believes that the good results reported in the treatment of the disease by drugs of the group are due to the elimination of superimposed bacterial infections. SPINING[11] found that fifteen adults so treated, having other associated acute eye infections, recovered rapidly as far as these secondary infections were concerned, but the trachoma was practically unaffected. FORSTER[12] has reported favourably. He treated 167 American Indian school-children with sulphanilamide and found the disease to be arrested in 125 after a course of three weeks. In the remaining 42 a further course of the drug was required. Approximately fifty per cent. of the children in this school were trachomatous. THYGESON[13] treated 31 cases; 16 were cured, 11 ameliorated and 4 unchanged. Contrary to the usual experience the treatment did not appear to affect any associated mixed infections. Corneal lesions responded more rapidly than conjunctival ones, and papillary hypertrophy was more quickly

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[7] JULIANELLE (L. A.) & SMITH (J. E.). Studies on the Infectivity of Trachoma. IX. Immunological Aspects of the Rickettsial Concept.—*Amer. Jl. Ophthalm.* 1940. Jan. Vol. 23. No. 1. pp. 62–67. [14 refs.]

[8] JULIANELLE (L. A.). Studies on the Infectivity of Trachoma. X. Frequency and Distribution of the Inclusion Body and its Possible Relation to Pathogenesis.—*Amer. Jl. Ophthalm.* 1940. June. Vol. 23. No. 6. pp. 633–644. With 3 figs.

[9] REIS (Karolina). La réaction sérologique de Weil-Félix et le trachome.—*Rev. Internat. du Trachome.* 1939. Oct. Vol. 16. No. 4. pp. 181–192. [17 refs.]

[10] MACCALLAN (A. F.). Sulphonamide Treatment of Bacterial and Trachomatous Conjunctivitis.—*Rev. Internat. du Trachome.* 1939. Oct. Vol. 16. No. 4. pp. 197–201.

[11] SPINING (W. D.). Some Observations on the Use of Sulfanilamide in Trachoma and Associated Ocular Conditions.—*Amer. Jl. Ophthalm.* 1940. Mar. Vol. 23. No. 3. pp. 271–274.

[12] FORSTER (Wesley G.). Treatment of Trachoma with Sulfanilamide.—*Amer. Jl. Ophthalm.* 1940. May. Vol. 23. No. 5. pp. 532–534.

[13] THYGESON (Phillips). The Treatment of Trachoma with Sulfanilamide. A Report of 31 Cases.—*Amer. Jl. Ophthalm.* 1940. June. Vol. 23. No. 6. pp. 679–685. With 2 figs. [19 refs.]

reduced than follicular. WILSON[14] reports encouraging results with respect to corneal lesions, but the palpebral lesions were less responsive. BURNET, CUÉNOD & NATAF[15] have made further reports on their success with sulphonamide 33 [see this *Bulletin*, 1940, Vol. 37, p. 448]. This drug is a salt of K and Na with parasulphamide phenyl azosalicylic acid. It appears to be relatively non-toxic and to give such rapid relief to corneal lesions with their associated painful symptoms that patients readily come for treatment. From 3 to 4 gm. daily in regularly spaced doses of 0·5 gm. is administered by the mouth. It is claimed that a clinical cure is always obtained within a comparatively short time and that the patient is enabled to resume his ordinary occupation.

Three cases of unilateral trachoma are reported by AYBERK[16]. He suggests that some difference in its conjunctival epithelium may account for the apparent immunity of the healthy eye. It is noteworthy that all the affected eyes reported on had suffered from previous troubles—lachrymal in two cases and uveal in the third. [The reviewer's experience of unilateral trachoma has led him always to suspect the diagnosis. Dramatic cures may often be effected by suitable nasal, oro-pharyngeal or constitutional treatment with the relief of any existing refraction error.]

Cataract.—PHILPS[17] has investigated the source of the bleeding into the anterior chamber sometimes met with after cataract extraction. Haemorrhage occurred in 13·13 per cent. of 374 extractions in certain London hospitals. The wound was the source in 85 per cent. of these and in the remainder the bleeding came from the iris or from the conjunctival flap. Local trauma such as a squeeze of the lids leads to a rupture of the young vessels in the wound. Cases in which a corneo-scleral suture was used escaped the complication. Erythrocytes are unaffected by the aqueous humour but are absorbed by the iris.

Quinine Poisoning.—NASHED[18] observed a case of *quinine amblyopia* in a woman, aged 60, five days after she had swallowed a tablespoonful of quinine bihydrochloride in mistake for Epsom salts. Nothing strikingly abnormal was visible on ophthalmoscopic examination, but there was a slight haziness of the optic discs with narrowing of the retinal arteries and dilatation of the veins. The pupils were fixed and three-quarters dilated. Vision was reduced to recognition of hand movements. Pallor of the retinal reflex and the discs and a thread-like appearance of the arteries were noted four days later. After the expiration of five months vision could be corrected to 6/9, but the visual fields showed marked concentric contraction. Nashid favours the view that the visual failure is due to the toxic influence of the drug on the retinal nerve elements rather than on the local vascular system. In a discussion of this paper IBRAHIM describes a similar case in a man, aged 30,

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[14] WILSON (Rowland P.). A Preliminary Report on the Treatment of Trachoma with Sulphonamide Compounds.—*Bull. Ophthalm. Soc. Egypt.* 1939. Vol. 32. pp. 79–98.

[15] BURNET (Et.), CUÉNOD (A.), NATAF (R.) & ROUSSEL (H.). Traitement chronique du trachome par l'azoique sulfamidé 33.—*Arch. Inst. Pasteur de Tunis.* 1940. Mar. Vol. 29. No. 1. pp. 66–101. [22 refs.]

[16] AYBERK (Nuri F.). A propos de trois cas de trachome unilatéral.—*Rev. Internat. du Trachome.* 1939. Oct. Vol. 16. No. 4. pp. 192–197.

[17] PHILPS (A. Seymour). Post-Cataract Hyphaema.—*Brit. Jl. Ophthalm.* 1940. Mar. Vol. 24. No. 3. pp. 122–135. With 7 figs.

[18] NASHED (Elia). Quinine Amblyopia.—*Bull. Ophthalm. Soc. Egypt.* 1939. Vol. 32. pp. 209–221. With 2 figs. [19 refs.]

seen two hours after taking quinine sulphate. Retinal oedema, hazy discs and contracted arteries were noted. Vision improved to 6/18 within a fortnight.

An interesting description of the development of ophthalmology in Bombay has been furnished by DUGGAN & CHITNIS[19]. The General Hospital was first established in Bombay in 1676, some twelve years later than that in Madras, but separate provision for treatment of diseases of the eye was not made until 1823. The Grant Medical College was completed in 1845, and, eighteen years later, owing to the generosity of COWASJI JEHANGIR, the ophthalmic institution bearing his name was erected in connexion with it. Accommodation for in-patients has grown from 40 beds to 100. The Hospital and School have now acquired an international reputation through the care and skill of the past and present Superintendents and Staff. Sir Jamsetjee Duggan has been in charge for the past eighteen years.

H. Kirkpatrick.

BELLECCI (Paolo). La reazione di Weil Felix nei Libici tracomatosi. [**The Weil-Felix Reaction in Trachoma in Libya.**]—*Ann. di Med. Nav. e Colon.* 1940. Jan.–Feb. Vol. 46. No. 1–2. pp. 26–33.

The author summarizes his paper as follows :—

" The author . . . . makes reference to the results of his researches on the power of the serum of patients with trachoma in Libya to agglutinate *Proteus X19*. In 67 patients, 25 of whom had cicatrizing lesions, 29 subacute and 13 florid lesions, agglutination to *Proteus OX19* was observed in only 3 instances, to a titre of not more than 1 : 50, and to *Proteus OXK* in one instance, to a titre of 1 : 100.

" He considers that with the strains of Proteus so far employed the Weil-Felix reaction has no practical or theoretical value in elucidating the aetiology of trachoma, since in this disease it is not intensely and constantly positive as it is in typhus."

C. W.

[19] DUGGAN (J. N.) & CHITNIS (V. K.). The Development of Ophthalmology in Bombay.—*Brit. Jl. Ophthalm.* 1940. May. Vol. 24. No. 5. pp. 213–229.

MISCELLANEOUS.

FIELD (J. W.). **A Simple and Rapid Method of staining Malarial Parasites in Thick Blood Smears.**—*Trans. Roy. Soc. Trop. Med. & Hyg.* 1940. Aug. 16. Vol. 34. No. 2. pp. 195–202. With 1 plate.

The observations of PAMPANA [this *Bulletin*, 1939, Vol. 36, p. 410] and of SIMONS [*ibid.*, 1938, Vol. 35, p. 814] show that freedom from distortion of protozoa and leucocytes in thick blood smears may best be obtained by the use of basic stains in isotonic solution. The author has based his experiments on this assumption. Essential conditions appear to be : Limitation of staining to about one second, buffering of the stain to *p*H 6·6 to 7·0, at which contrast is maximal, and the retention of some of the haemoglobin to provide a pale yellow background.

Of the strains tested, brilliant cresyl blue is the most generally satisfactory, and the composition recommended is as follows :—

Brilliant cresyl blue	1·0 gm.
Disodium hydrogen phosphate (anhydrous) ...	1·0 gm.
Potassium dihydrogen phosphate (anhydrous)	1·25 gm.
Distilled water	100 cc.

The phosphate salts are first dissolved, giving a solution approximately isotonic with serum and of *p*H 6·6. The stain is added and after filtration is ready for use. An alternative stain consists of :—

Methylene blue	0·4 gm.
Eosin	0·4 gm.
Isotonic phosphate solution	100 cc.

The methylene blue is dissolved in 80 cc. of the phosphate solution, the eosin in the remaining 20 cc., and the eosin solution is then added to the methylene blue and the whole is filtered. With these stains any formation of scum is an indication for further filtration.

The blood smears should be not more than 50μ thick, at which the fingers of a watch can be seen dimly through the dried film. The smear is dried rapidly by waving in the air, and is lightly fixed by passing through a flame for one second, but the slide should not be heated to such an extent that it cannot be held against the back of the hand. The slide is dipped in the stain for 1 second, immediately removed, and rinsed for 5 seconds by waving gently in a vessel of clean tap water ; it is then dried in the vertical position. Haemoglobin continues to drain from the smear during drying and leaves four distinct zones, depending on the amount of haemoglobin left behind :— A. at the upper thin edge, little haemoglobin, minute parasites such as young *P. falciparum* rings are best sought here ; B. adjoining A, the background is pale yellow, most parasites are best seen here ; C. central, containing too much haemoglobin ; D. at the lower thin edge, the parasites and leucocytes are often well contrasted against the smooth yellow ground.

By this method, correct diagnosis of the species of parasite may be made, in a very high proportion of cases, within 1½ minutes of first preparation of the smear, and in comparison with a thin smear there is approximately a sixteen-fold concentration of parasites. The author gives an account of the morphology of parasites stained by this method, pointing out that while the advantage of speed is very great, the disadvantages are so slight that diagnosis should seldom be in doubt.

C. W.

ROBERTS (J. I.). **Field's Rapid Method of staining Malaria Parasites.**—*East African Med. Jl.* 1940. Sept. Vol. 17. No. 6. pp. 232–234.

MICHELSON (Louis) & WILCOX (Aimee). **A Rapid Thick Film Blood Stain.**—*Public Health Rep.* 1940. July 5. Vol. 55. No. 27. pp. 1221–1222.

The stain is prepared as follows :—

"*A. Preparation of Wright-Giemsa Solution from Giemsa Powder and Wright's Solution.*

"Place 100 cc. of glycerine, C.P., anhydrous, in a bottle of 1 liter capacity which has a tightly-fitted screw cap or stopper. Weigh accurately

1·515 gm. Giemsa powder (National Aniline Dye Co., N Ge-3) and suspend in glycerine. Fit stopper tightly, cover entire bottle neck with a double thickness of wrapping paper, and secure with large elastic bands. (These precautions are taken to prevent moisture from being absorbed by the Giemsa-glycerine mixture during the heating period in the water bath.)

"Heat the bottle of Giemsa-glycerine mixture in the water bath at 55°–60° C. for 2 hours, mixing well with a glass stirring rod at half-hour intervals. At each stirring, remove bottle from water bath. After 2 hours, remove from water bath and allow to cool. Then add 100 cc. of unfiltered Wright's Stain Solution (B) to the bottle of Giemsa-glycerine solution. Mix well by vigorous shaking and let stand overnight. On the next morning add 800 cc. of unfiltered Wright's Stain Solution (B) to the above mixture. Shake vigorously. Filter into a small bottle the amount of stain needed for a few day's staining. Stain requires no aging and can be used immediately.

"*B. Preparation of Wright's Stain Solution from Powder (National Aniline Dye Co.).*

"Place 1,000 cc. of methyl alcohol, acetone free, neutral, and preferably redistilled, in a bottle of 1 liter capacity which has a tightly-fitted screw cap or stopper. Weigh accurately 2 gm. Wright's powder and dissolve in the methyl alcohol. Wrap the bottle in paper and store in a dark place protected from ammonia fumes for at least 1 month. At frequent intervals while stain is aging shake bottle vigorously. At the end of 1 month, test for staining properties. The Wright's Stain Solution must give satisfactory blood-cell staining before it can be used in the preparation of this Wright-Giemsa stain.

"IMPORTANT.—All ingredients must be of reagent quality, preferably from freshly opened bottles. All glassware must be chemically clean and perfectly dry."

The stain is diluted 1 in 10 with distilled water buffered to *p*H 7·0. Thick films are stained for 10 minutes and washed in distilled water for one minute. Thin films may be stained in a similar way but should first be fixed in methyl alcohol and the stain should be poured over them while they are still wet with alcohol. A prolonged method, in which the stain is diluted 1 in 40 and staining is allowed to proceed for 45 minutes, may also be used. *C. W.*

ROE (M. A.), LILLIE (R. D.) & WILCOX (A.). **American Azures in the Preparation of Satisfactory Giemsa Stains for Malaria Parasites.**—*Public Health Rep.* 1940. July 12. Vol. 55. No. 28. pp. 1272–1278.

The supply of German stains to America has been interrupted, and the substitution of azure I or azure A (which have been considered identical) of American manufacture, for the azure I of Grübler, has not yielded good results in the preparation of the Giemsa stain. The authors have therefore examined individually the staining actions, on malaria parasites, of the possible chemical constituents of Grübler's azure I, made up in simple combinations with eosin. These constituents belong to the group of thiazin dyes, namely, methylene blue, azure B, azure A, azure C, thionin and certain others. Methylene blue stains the cytoplasm well, but not the chromatin; azure B gives excellent results; azure A is not so good and the others are poor. Azure B with methylene blue in proportion of 1 : 1 to 1 : 4, with appropriate amounts of eosin, with or without a small quantity of azure A, give

very satisfactory results. The formulae of three satisfactory solutions are given, of which perhaps the best is :—

	Pure dye weight	*Total dye weight*
Azure B (estimated) 80 per cent. ...	200 mgm	250 mgm
Azure A 90 per cent.	50 ,,	55 ,,
Methylene Blue 87 per cent. ...	270 ,,	310 ,,
Eosin Y 93 per cent.	500 ,,	537 ,,
Glycerin	50 cc.	50 cc.
Methyl alcohol	50 ,,	50 ,,

[The pure dye weight of Azure A is given in the original as 500 mgm. Evidence in the text shows this to be a misprint, the correct amount is 50 mgm. as stated above.]

It is pointed out that the proper synonym for German azure I is azure B, not azure A, as has been supposed. *C. W.*

SCHRETZENMAYR (A.). **Sternal Puncture in Malaria and Other Tropical Diseases.**—*Far Eastern Assoc. Trop. Med. C.-R. Dixième Congrès, Hanoi, 26 Nov.–2 Dec., 1938.* Vol. 2. pp. 99–110. With 6 figs. on 3 plates.

The claim is made by the author that sternal puncture is a simple procedure which may furnish very useful information and should be used as a routine diagnostic measure by those practising in the Far East. In malaria, for example, he states that in 27 patients suspected on clinical grounds he found confirmatory evidence from examination of the marrow of 19, although blood smears had revealed no parasites ; in districts where kala azar occurs there are many cases " which can only be detected by this means " [but see this *Bulletin*, 1939, Vol. 36, p. 1030 ; 1940, Vol. 37, p. 349] ; examination of the marrow in cases of hookworm anaemia will tell us when there is regeneration going on or whether the condition is aplastic, for, if the latter, the prognosis is very grave. In South China acute leukaemia is liable to be confused with haemorrhagic smallpox ; examination of the sternal marrow will show in the latter a distinct increase in reticulo-endothelial cells—a point of much diagnostic value. *H. H. S.*

TURNER (Olga). **Notes on the Preparation of Certain Medicinal Solutions for Intravenous Injections and Enemata used in the Tropics.**—*Trans. Roy. Soc. Trop. Med. & Hyg.* 1940. June 27. Vol. 34. No. 1. pp. 109–113.

This is a useful paper. It is pointed out that drugs for intravenous use which are issued in solid form should be used on the day on which they are dissolved in sterile distilled water ; the solutions should never be left to stand overnight. Pyrogen-free distilled water should be freshly prepared, preferably on the day of use, in a pyrex all-glass apparatus, of which details are given.

Antimony sodium tartrate is less toxic than the potassium salt. It can be sterilized by boiling without harm, but must be freshly prepared. Quinine dihydrochloride solution can also be sterilized by boiling or in the autoclave ; it should not be stronger than 1 grain in 1 cc. Sodium bicarbonate solution must not be sterilized by heat as this converts the bicarbonate to the toxic carbonate ; the chemically

pure salt should be added to fresh, cooled, double distilled water. Sodium citrate solution may be sterilized by heat.

It is pointed out that in preparing yatren enemata tap water must not be used and the vessels for storage must not contain any trace of acid. Solutions of yatren must not be boiled, as this causes decomposition. Cod liver oil enemata should be prepared as emulsions with gum acacia (not with the irritating tincture of quillaia) but in preparing bismuth subgallate in olive oil or cod liver oil no emulsifying agent must be used; instead, thorough grinding in a mortar during preparation, and thorough stirring with a suitable rod immediately before use, are essential. *C. W.*

SICÉ (A.). Le nourrisson européen sous le climat du Soudan français. [**European Infants and the Climate of the French Sudan.**]—*Bull. Soc. Path. Exot.* 1940. Mar. 13. Vol. 33. No. 3. pp. 156–159.

The climate of the French Sudan may be divided into a hot dry season from February to May, a rainy season from June to October, and a cool season from November to January. The period dangerous to infants is the hot dry season, when humidity is at its lowest (17 to 28 per cent. daily average) and the temperature rises to 40 or 41°C. The heat regulating mechanism of infants is not so efficient as that of adults, and the body temperature of infants therefore varies considerably with that of the surrounding atmosphere. Particularly marked is the evaporation of fluid from the skin and mucous membranes, and in this connexion evaporation from the lungs is emphasized. The factors leading to heat stroke are therefore present, and this may take certain forms:—(1) slight, with vomiting and diarrhoea; (2) grave, with restlessness, rapid pulse and respiration, high temperature and sometimes meningism; (3) very grave, with sudden onset leading to collapse and cardiac failure.

Remarking that many houses have been built on unsound lines, with neglect of the provision of verandahs and consequent overheating of the buildings, the author points out the importance of raising the indoor humidity by means of bowls of water or even fine garden nebulizer sprays. In addition he emphasizes the necessity for providing drinking fluid to infants at the rate of 180 gm. per kgm. body weight each 24 hours during the first month of life. This is diminished to 122 gm. in the second month, whereas for an adult 40 gm. is enough in ordinary circumstances. Sweetened boiled water, with or without fruit juice, is recommended. In European infants thus treated, and in whom wise dietetic measures are taken, the author has seen perfect health maintained. He has, however, seen infants not so well protected die of heat stroke. *C. W.*

HASSELMANN-KAHLERT (Margaret). Ueber Menstruationsstörungen bei der gesunden weissen Frau in den Tropen. [**Menstrual Disturbances in Healthy White Women in the Tropics.**]—*Arch. f. Schiffs- u. Trop.-Hyg.* 1940. Mar. Vol. 44. No. 3. pp. 124–133. [23 refs.]

In the Philippine Islands menstruation in white girls commences usually between the ages of 9 and 12 years, in native girls between 11 and 14. The commonest menstrual disturbance of healthy women in Manila is oligo-amenorrhoea [presumably this means oligomenorrhoea

or amenorrhoea], and this is more often found in white women than in Chinese, Japanese or natives. Manila is free from malaria and the question of the effect of quinine on menstruation does not therefore arise, neither is there any question of malnutrition. Of 251 white women 30 per cent. had oligo-amenorrhoea, and in no case was there any genital or other disease to account for it. Normal menstruation was re-established on return to a temperate climate.

Associated symptoms include a sensation of fulness in the pelvis, headache and even symptoms suggesting heatstroke ; there is a strong psychological element, and symptoms usually associated with the menopause may occur in young women. To exclude pregnancy the author uses her own modification of the Cuboni test.

In treatment the author has used Agomensin and more recently Progynon B. These are given during the period 14 to 10 days before the calculated time at which menstruation should occur and are continued in this way for 4 or 5 months. The author claims (without, however, giving details of her results in the series) that this does not seem to be a question of substitution therapy, but rather a true causal therapy. With Theelin, Progynon, Unden, Folliculin and Estrogen the results have not been satisfactory, and vitamin and thyroid medication was unsuccessful.

Oligo-amenorrhoea does not appear to influence fertility, and sterility may, of course, occur in those with normal menstruation ; in these vitamin E therapy may be useful.

The author considers that the climate of the tropics exerts an influence on the ovaries and pituitary, and advises that women who show menstrual disorders, particularly oligo-amenorrhoea, in temperate climates should not live in the tropics for long periods, but these menstrual disorders do not appear to lead to any permanent damage.

C. W.

Lee (Douglas H. K.) & Courtice (R.). **Assessment of Tropical Climates in Relation to Human Habitation.**—*Trans. Roy. Soc. Trop. Med. & Hyg.* 1940. Apr. 30. Vol. 33. No. 6. pp. 601–614. With 6 figs. (1 map on plate). [11 refs.]

This paper deals with the assessment of climate from the thermal standpoint. It is recalled that the environmental factors which affect the loss of body heat and the maintenance of thermal equilibrium within the body are the temperature, humidity and rate of movement of the air and the radiation from the surroundings. A scale of warmth which combines the first three of these in a single index, but neglects radiation, is effective temperature.*

It is remarked that in a large land area passing from temperate to tropical regions any climatic index should show a high degree of correlation with latitude when corrections have been made for altitude. The authors have calculated the effective temperatures (assuming still air) for 9 a.m. in the hottest months of the year, the coldest months, and the averages for the year, for each of the recording stations in Queensland and the Northern Territory of Australia, and have corrected these figures as well as the dry bulb temperatures for altitude. They have then correlated each of these against latitude. Separate

* The effective temperature of an atmosphere connotes the temperature of still air saturated with moisture in which the subjects of certain American experiments experienced the same feeling of warmth as in the atmosphere in question.

correlations have been computed for the humid coastal regions and for the arid inland regions. Both sets of correlations are high and it is therefore concluded that effective temperature is a good measure of climatic warmth, when radiation is neglected. It is also concluded that effective temperature correlates more highly than dry bulb temperature with latitude, and is therefore a better measure of climate.

Isotherms for corrected effective temperature in the hottest month do not run roughly parallel with latitude, but take a sudden northward trend as the humid coast is approached. Hence it is inferred that as an index of climate at high temperatures the effective temperature scale lays undue stress on the dry bulb temperature.

A comparison is made of the climate of twelve Queensland towns. It is remarked that regard should be paid to the extent in both duration and degree to which the effective temperature rises above the comfort zone (the zone used is that which is accepted as applying to American subjects in American conditions), and to the magnitude of seasonal and diurnal variations. The authors had not the data necessary for an exhaustive analysis and, admittedly, had to satisfy themselves with a semi-quantitative survey. Nevertheless they express their results quantitatively and base their comparison on the number of day-degrees (or degree-days) per year above or below the comfort zone. Thus it appears that, taking the 9 a.m. figures, the gross number of day-degrees above the upper limit of the comfort zone at Cape York is 1570, while the number below the lower limit is nil; hence the net excess is 1570 day-degrees. At Rockhampton the gross numbers of day-degrees above and below the comfort zone are 160 and 290 respectively, so that the net excess is –130 day-degrees. Sub-tropics are taken to mean places where the 9 a.m. figures give no net excess above the comfort zone. Marginal tropics include places where there is a 9 a.m. net excess, but where the 3 p.m. gross excess does not exceed 1,200 day-degrees. Tropics include places where the gross 3 p.m. excess exceeds 1,200 day-degrees. Subdivisions suggested for the tropics are (*a*) places permissible for Caucasian habitation but requiring selection and marked adaptation, where the 9 a.m. effective temperature does not exceed 80°F.; and (*b*) places not suitable for continuous Caucasian habitation, where the 9 a.m. effective temperature exceeds 80°F. On the suggested basis Rockhampton, Bundaberg, Brisbane, Longreach and Charleville are classed as sub-tropical, while the marginal tropics include Mackay and Townsville. Of the tropical towns Cardwell, Cairns and Cloncurry are in group (*a*), while Cape York and Burketown fall just inside group (*b*).

[Messrs. Lee and Courtice have produced an interesting paper, but certain criticisms arise. We are not told how the standard errors or probable errors of their correlation coefficients are calculated, but, it is very doubtful whether in any case the correlations of latitude with effective temperature and dry bulb temperature differ significantly, and in some cases the correlations with dry bulb temperature are the higher. Yet it is claimed, unjustifiably as it would appear, that the results show effective temperature to be the better index.

The northward trend of the isotherms is assumed to indicate some failure of effective temperature as a measure of warmth. This criticism needs more substantiation than is given. It appears that when the records for inland and coastal stations are grouped together the correlation of effective temperature with latitude is not very good and therefore effective temperature is not an accurate index of

latitude, but this does not justify the assumption that it does not measure environmental warmth as it affects man.

In the comparison of Queensland climates it is not stated how the numbers of day-degrees of temperature excess above the comfort zone are computed, but they appear to have been calculated from the mean monthly temperatures. If that is so the figures for some stations may be subject to substantial error. It can readily be shown that if on some days during the month the temperature is below the upper limit of the comfort zone, the true number of day-degrees excess for that month will be under-estimated if the calculation is made from the mean temperature for the month. It is arguable that the variations on either side of a comfort line (*e.g.*, the upper limit of the comfort zone, or the mid-line of the comfort zone) might have furnished a better basis for the assessment of climate than that chosen by the authors. On this basis more satisfactory values would have been obtained from monthly averages.] *T. Bedford.*

NOOSTEN (H. H.). Le " Rouget " chez les enfants à Bali. [**"Rouget" in Bali Children.**]—*Far Eastern Assoc. Trop. Med. C.-R. Dixième Congrès Hanoi, 26 Nov.–2 Dec., 1938.* Vol. 2. pp. 79–83. With 3 figs. on 1 plate. [10 refs.]

The author writes of a condition which he has seen recently in two children, but which he believes to be far from rare—resulting from the bite of a larval Trombicula, known locally as Tungu. [There are several Balis ; in Belgian Congo, in Nigeria, in the Cameroons, and an island in the Dutch East Indies. Probably, it is the last to which this paper refers.] Clinically, the chief symptom is swelling of the genitalia and is seen in young children of 3 or 4 years of age who play, naked, round the edges of wells. In the moss on the stones at the well-mouth is a small red insect which bites the children, and in some cases, it is said, may be found embedded in the swollen tissues. Swelling and itching appear two or three days after the insect bites. " Rouget " occurs in Java, Sumatra, and in fact is said to be fairly well known to the natives throughout the Netherlands Indies.

H. H. S.

MCKAY (W. J. Stewart). **Tick Bite and Allergy.** [Correspondence.]—*Med. Jl. Australia.* 1940. Mar. 30. 27th Year. Vol. 1. No. 13. pp. 458–459.

The writer of this letter discusses the bite of the dog tick *Ixodes holocyclus*, the effects of which he claims to be due to allergens injected in the venom. In dogs and children are produced symptoms of vagotonia (" spasm or paresis of the muscles of the gullet "). This explains the fact that dogs will not drink at this stage. Children may also show sympathicotonia in the form of diarrhoea. The author believes that violent reactions to tick bite are not due to some general body substances in the ticks, but are caused by the venom which is produced in the salivary glands. A guineapig to which one tick becomes attached shows no symptoms until the fourth day ; on that day the tick takes in a large amount of blood and the salivary glands increase in size. On the fifth day more venom is injected and the guineapig dies the same night. " It almost looks as if the first injections given

sensitized the guineapig, and the injections on the fifth day killed it by anaphylactic shock." In man urticaria may be produced within a few hours of the bite. *C. W.*

BOESOIRIE (Chasan). Vischvergiftiging door een ikan nogi-nogi. [**Fish Poisoning by Nogi-nogi** (*Tetrodon argenteus*).]—*Geneesk. Tijdschr. v. Nederl.-Indie.* 1940. May 21. Vol. 80. No. 21. pp. 1338–1340. [Summary appears also in *Bulletin of Hygiene.*]

The fish which caused the poisoning was identified as *Tetrodon argenteus*. It, together with other fish, was cooked and eaten by nine persons, 4 adults and 5 children. Those who partook of the other fish were slightly affected, owing, it is stated, to poison carried over from the Tetrodon. One adult and one child died within two hours of the onset of symptoms. The symptoms detailed in the case of one man who was seriously ill but recovered are described. He complained of a sensation of swelling of lips and tongue, making speech difficult; there was much salivation and his limbs "went to sleep" after about two hours; there was a sensation of burning in the stomach, his breathing grew laboured and he was cyanosed and drowsiness increased with tremors. Coramin was administered. He felt drowsy for a week, the tongue symptoms persisted for two days. The poison seems to be present in the mucus which covers the fish, and it has a neurotoxic action. [The post-mortem appearances are not recorded.] *H. H. S.*

SMITH (Charles Edward). **Epidemiology of Acute Coccidioidomycosis with Erythema Nodosum ("San Joaquin" or "Valley Fever").**—*Amer. Jl. Public Health.* 1940. June. Vol. 30. No. 6. pp. 600–611. With 4 figs. [27 refs.]

This is a careful epidemiological study of what is known as San Joaquin fever, or by its synonyms Valley fever, Desert fever and Desert rheumatism [see this *Bulletin*, 1939, Vol. 36, pp. 507–8]. The disease usually presents an influenza-like initial phase, and after an interval, which may be as short as two or as long as eighteen days, an eruptive phase of erythema, nodosum or multiforme, with at times an arthritis and phlyctenular conjunctivitis. In one to three weeks the rash fades but the affected areas may remain pigmented for months. No fatal case is recorded. In 1936 it was shown to be due to *Coccidioides immitis*. The incidence is greatest in the counties of Tulare and Kern, California, and in a period of 17 months, December 1937–May 1939, 432 cases were observed. The organism may be found in the sputum (but many patients have ceased to expectorate by the time they come for treatment) and proved by culture and by animal inoculation. Some four-fifths of the patients do not present themselves until the rash appears. The period of incubation ranges between one and three weeks, most commonly two weeks. Sensitivity to coccidioidin is established usually within a fortnight of the onset of symptoms and may be as early as two days or as late as seventeen. The allergy after attack is of long duration; one case is mentioned in which a very strong reaction followed the injection of 0·1 mgm. coccidioidin though 9 years had elapsed since the known infection, and only two of the 432 patients were said to have had a previous attack and one of these is not absolutely certain.

Infection is generally acquired by inhalation of the fungus spores and it does not pass from person to person. Instances are given in

which sharing a bed with a patient did not result in transmission of infection ; in other words, " the endosporulating spherules of Coccidioides which occur within the animal host and are found in the sputum are rarely if ever infectious. Responsibility lies with the chlamydospores characteristic of the fungus in nature and readily adapted to widespread dissemination." As regards seasonal prevalence, fewest cases occurred in the wet season with little field work ; in the hot summer and autumn irrigation ceases, dust is troublesome and cases increase. For example, of 344 cases in 1938, 73 occurred in the first six months and then in successive months 36, 52, 41, 65, 43, 34, or 271 in the latter half of the year. In January 1939 there were only 11 and in January 1938, 10. Social and economic conditions seemed to play no part ; newcomers are mostly attacked, half the patients had been in the Valley less than a year and two-thirds less than two years. Tests with coccidioidin show that more than half of 2,718 school-children in Kern county schools reacted positively and the percentage rose progressively from 17 among those who had resided in the county for less than one year to 77 among those of 10 years' residence.

The influenza-like onset may lead to erroneous diagnoses of influenza, bronchopneumonia, tuberculosis and others and the rash to measles or smallpox. Only some five per cent. of those infected develop erythema nodosum, so the 432 patients probably represented 8,000 or more new infections. *H. H. S.*

PROMMAS (C.). **Rhinosporidial Infection in Siam. (Report of Two Cases.)**—*Far Eastern Assoc. Trop. Med. C.-R. Dixième Congrès Hanoi, 26 Nov.–2 Dec., 1938.* Vol. 2. pp. 135–139.

Two cases are recorded, both in Siamese farmers, aged 20 and 21 years, in different districts in Siam. Surgical removal resulted in their leaving hospital in good health. Though not uncommon in India, the disease is rare in Siam, these being the only two cases seen in the course of 13 years' work at the Siriraj Hospital. It is for this reason that it was thought to be worth putting on record. *H. H. S.*

RAM (Tehl). **Prontosil Album in Gangrenous Stomatitis.**—*Indian Med. Gaz.* 1940. Mar. Vol. 75. No. 3. p. 162.

The patient, a boy of 10, developed gangrenous stomatitis while in hospital with symptoms resembling dysentery. Sloughing and foul ulcers were found on both upper and lower gums and glossy oedema of the cheeks was present. Treatment with pure nitric acid locally and mouth washes was given and 4 tablets [presumably each of 0·5 gm.] prontosil album were given daily to a total of 14. Within a week the gums were healthy, a remarkable result in a condition which in the author's experience in the past had invariably proved fatal.

C. W.

TROWELL (H. C.). **The Recognition of Nutritional (Tropical) Macrocytic Anaemia in Uganda and its Previous Confusion with Malarial Splenomegaly and Splenic Anaemia.**—*East African Med. Jl.* 1940. Apr. & May. Vol. 17. Nos. 1 & 2. pp. 14–29 ; 60–70. With 1 fig. [21 refs.]

The author believes nutritional macrocytic anaemia to be relatively common although seldom diagnosed in E. Africa, and, after summarizing current views on nutritional anaemias and reviewing standard

methods of investigation, he suggests that the condition should be suspected in the following circumstances. The anaemia appears to be the primary disease and not to be due to cachexia or other causes, the red cell count is below three million per cmm., and the colour index, when the haemoglobin is estimated by the Sahli method, is over 0·7; this value for the colour index is taken because the Sahli method, commonly used in E. Africa and thought by the author to be best for the tropics, gives high readings of haemoglobin, 85 per cent. on the scale corresponding to 14·5 gm. haemoglobin per 100 cc. blood. Stained films show great inequality of cell size, few poikilocytes, no hypochromia, and increased polychromasia; there is often a neutropenia. Gross splenomegaly and fever are often present and intercurrent malarial infections are common. The estimation of the mean corpuscular volume is relatively simple and establishes the diagnosis. The mean corpuscular diameter, estimation of which by the Price-Jones curve is not a practical routine procedure and by any diffraction method valueless, is often within the normal range of European blood and may even be low compared with African standards, whereas the mean corpuscular volume is always increased as the cell thickness is greater than normal. The administration of iron causes little or no rise in the haemoglobin but the cases respond to injections of certain, but not all, preparations of liver or to large doses of marmite; liver by the mouth or in small doses parenterally yields no improvement.

Finally, the author suggests that haematological findings should be measured against European standards, as figures representing so-called normals obtained from observations on Africans may be influenced by minor degrees of deficiencies among the general population.

F. Murgatroyd.

ANDERSON (T. F.) & ROBERTS (J. I.). **Macrocytic Anaemias in Kenya. A Preliminary Report.**—*Trans. Roy. Soc. Trop. Med. & Hyg.* 1940. Apr. 30. Vol. 33. No. 6. pp. 615–622. With 8 figs. [11 refs.]

Of four cases of macrocytic anaemia in adults in Kenya only one had a complete achlorhydria and in this case the van den Bergh reaction was negative. The authors believe all were examples of nutritional anaemia, the diet of each patient being mainly carbohydrate with marked deficiency of animal protein. In two cases the exciting cause of the anaemia appeared to be pregnancy. In two patients hyperbilirubinaemia or jaundice suggested a haemolytic factor, possibly malaria, although the only patient in whom parasites were found had a negative van den Bergh reaction.

Animal protein, liver and marmite were given and all the patients rapidly improved excepting one who seemed to require a small blood transfusion (20 cc.)

A fifth case of macrocytic anaemia occurred in a breast-fed infant of 8 months. The condition differed from Lederer's acute haemolytic anaemia in the absence of icterus and reticulocytosis, and in the recovery without transfusion; it is suggested that the anaemia was due to dietary deficiency although the mother showed no signs of anaemia.

Target cells were found in four of the cases, including that of the infant.

F. Murgatroyd.

FAIRLEY (N. Hamilton). **Tropical Macrocytic Anaemia in an Indian treated with Anahaemin.**—*Lancet.* 1940. June 22. pp. 1118–1119. With 1 chart.

WILLS and EVANS stated that Indian cases of tropical macrocytic anaemia do not respond to highly purified extracts of liver, such as anahaemin, and suggested that crude extracts contain an additional haemopoietic factor, a view supported to some extent by NAPIER. [See this *Bulletin*, 1938, Vol. 35, p. 846, and 1939, Vol. 36, p. 935.] FOY and KONDI, using large doses of anahaemin, obtained a satisfactory response in Macedonian cases and thought that such cases differed from Indian cases.

The present observation concerns an Indian whose blood examination before treatment showed: erythrocytes 1,700,000 per cmm.; haemoglobin 38 per cent. (Haldane); colour index 1·1; mean corpuscular volume 105·9 cμ; mean corpuscular average thickness 2·16 μ; mean corpuscular diameter 7·91 μ; megalocytes 18·8 per cent.; reticulocytes 1·2 per cent.; leucocytes 10,000 per cmm.; and platelets 201,000 per cmm. Numerous Ehrlich megaloblasts were seen in smears of the bone marrow. The van den Bergh indirect reaction was only 0·5 unit, gastric analysis demonstrated the presence of hydrochloric acid after histamine injection, the glucose tolerance test fell within normal limits and faecal fat analysis gave no evidence of sprue. No parasites were seen in blood films, the liver and spleen were not enlarged, and no eggs or protozoa were found in the stools. The patient's diet had contained only about 4 ounces of fish or meat weekly, and the case was considered to be one of uncomplicated tropical macrocytic anaemia.

Anahaemin was given intramuscularly, 4 cc. daily for three days followed by 2 cc. daily for a further twenty-nine days, with rapid clinical improvement. There was a reticulocytosis of 16·4 per cent. on the eighth day followed by rapid regeneration of blood representing an average daily increase of 85,000 erythrocytes per cmm. and 1·5 per cent. haemoglobin. At the forty-third day blood examination showed: erythrocytes 4,710,000 per cmm.; haemoglobin 98 per cent.; colour index 1·0; mean corpuscular volume 92·5 cμ; mean corpuscular diameter 7·6 μ; and megalocytes 2·6 per cent.

Although the response was satisfactory the dosage of anahaemin was very large. The author states that this extract possesses no advantage over cruder extracts given parenterally and on the grounds of expense alone its administration is prohibitive in the routine treatment of tropical macrocytic anaemia. *F. Murgatroyd.*

SMART (John) [compiled by], with the assistance of other Members of the Staff of the Department of Entomology. **Insects.**—*British Museum (Natural History). Instructions for Collectors No. 4A.* pp. vi+164. With 42 figs. 1940. London: Printed by Order of the Trustees of the British Museum.

The author is to be congratulated on producing a new and valuable piece of work. Its purpose is to assist those who are collecting material for the British Museum, but it will be valued by collectors generally and also by those who maintain museums and reference collections in this country.

The introduction emphasizes the need for careful collecting and labelling and endeavours to explain what it is the British Museum

wants to receive. The author then gives a short general account of what is meant by an insect and a brief outline of the way in which these things live. He then passes on to "Notes on the orders of insects: how and where to collect them." His intention is to help the man in the field to recognize members of the different orders and even some of the more important families. The admirable line drawings will certainly assist, but one may doubt whether it is possible in so short a space to do anything of value. Classification is so large a subject and demands such a considerable knowledge of anatomy that it might perhaps have been thought better to refer the reader to one or two well-known books.

The remainder of the book is excellent. It consists of practical notes on methods of collecting, storing, posting, labelling, etc. It is full of useful well tried hints and tips; though it cannot in the nature of things be complete, it includes a number of rather specialized apparatuses such as Berlese's funnel and Williams's light trap.

P. A. Buxton.

JOHNSON (C. G.). **Development, Hatching and Mortality of the Eggs of *Cimex lectularius* L. (Hemiptera) in Relation to Climate, with Observations on the Effects of Preconditioning to Temperature.**—*Parasitology.* 1940. June. Vol. 32. No. 2. pp. 127–173. With 13 figs. [30 refs.]

This paper, which is part of a wider programme on the ecology of the bed-bug, contains a mass of experimental data on the relations of the eggs to climate. The following points have been selected as being of most general interest.

Room temperatures in England are often between 0° and 13°C. for many months at a time. Many of the experiments, directed towards finding the longest possible time an egg can survive and the lowest possible temperature at which it can hatch, have been made over this part of the temperature scale. Early embryonic development occurs within the female, so that eggs laid soon after a meal take longer to hatch than those laid later (9½–6 days at 23°C.). This time is not influenced by atmospheric humidity. When eggs are laid at 23°C. the lowest temperature at which complete development and hatching can occur is 13°C.; but eggs allowed to develop at this temperature or over can still hatch at 8°C.; and some development may occur as low as 4°C. At 13°C., complete development and hatching only takes place when the relative humidity is between 75–90 per cent. Humidities of 99–100 per cent. cause a high mortality. Eggs kept at 1–13°C. can survive some time and still hatch if the temperature is subsequently raised. They survive longer if the temperature is near 13°C., and it is estimated that the longest possible period of survival, which occurs at about 12°C., is nearly 80 days. *V. B. Wigglesworth.*

ZUMPT (F.). Ueber neuere Untersuchungen zur Rolle der Bettwanzen als Krankheitsüberträger. Mit einem Beitrag zur Terminologie der Krankheitsübertragung durch Tiere. [**Recent Investigations into the Rôle of the Bed Bug in the Transmission of Disease.**]—*Zent. f. Bakt.* I. Abt. Referate. 1940. Apr. 11. Vol. 136. No. 19/20. pp. 401–414. [63 refs.]

MACFIE (J. W. S.). **The Genera of Ceratopogonidae.**—*Ann. Trop. Med. & Parasit.* 1940. Apr. 30. Vol. 34. No. 1. pp. 13–30.

ZAVATTARI (Edoardo). L'aeroplano quale trasportatore di insetti vettori o attori di malattia. [**Transport of Insect Disease Vectors by Aeroplane.**]—*Riv. di Biol. Colon.* Rome. 1939. Dec. Vol. 2. No. 6. pp. 425–429. English summary (4 lines).

"The author basing himself on the observations of Sarel WHITFIELD made in the Khartoum Airport reports the possibility of transport of insect vectors and agents of disease into Italy by air. He proposes some researches in the Airport of Littorio in Rome in order to verify the importance and extent of such transports."

Scabies : Notes on a New Method of Treatment. Bwele : Inzila Impya Ya Ku Silika. 1 p. leaflet. Revised. [Sent to the Bureau by the D.M.S., Northern Rhodesia.]

The original leaflet noticed in this *Bulletin*, 1940, Vol. 37, p. 230, contained an error in stating that after boiling 3 lbs. of slaked lime and 3 lbs. of sulphur together in a 4 gallon petrol tin filled with water the sediment should be discarded and the clear liquid used as a paint for the treatment of scabies. It should have stated that before use both the sediment and the clear liquid should be well stirred together and that the mixture should be used. In sending a copy of the revised leaflet to the Bureau, the Director of Medical Services, Northern Rhodesia, writes that this method of treating scabies has been developed in connexion with what are known locally as "itch campaigns" and that these campaigns, which are the beginning of medical work undertaken by the Native Authorities on behalf of their own people, have proved very popular and on the whole very successful.

R. L. S.

TALIAFERRO (William H.). **The Mechanism of Immunity to Metazoan Parasites.**—*Amer. Jl. Trop. Med.* 1940. Mar. Vol. 20. No. 2. pp. 169–182. [16 refs.]

KIRKLAND (W. B.). **Endemic and Epidemic Diseases in the Northern Territory.**—*Health.* Canberra. 1939. Nov. Vol. 17. No. 11. pp. 121–127.

REVIEWS AND NOTICES.

CANTON (N. A.) [M.B., D.P.H., Municipal Health Officer], ROSEDALE (J. L.) [Ph.D., D.Sc., F.I.C., Professor of Biochemistry, King Edward VII College of Medicine] & MORRIS (J. P.) [Assistant in Biochemistry, King Edward VII College of Medicine]. **Chemical Analyses of the Foods of Singapore.**—191 pp. 1940. Singapore : S.S. Government Publications Bureau, General Post Office, Fullerton Building. [$4 or 9s. 4d.] [Review appears also in *Bulletin of Hygiene.*]

The work of Professor Rosedale on the analyses and nutritive values of Malayan foods is well known to readers of this *Bulletin*

[see *Bull. of Hyg.*, 1935, Vol. 10, pp. 520, 587 ; 1936, Vol. 11, p. 572 ; 1937, Vol. 12, p. 215]. The present is a much more ambitious undertaking and must have entailed an enormous amount of work which, after a time, must have become a tedious, almost heartbreaking routine, but whose results will be of very great value for reference in many other parts of the world than Singapore.

The authors have analysed every available food and mixture of foods obtainable from hawkers, stalls, and eating houses in Singapore, and they believe that they have missed none. From the numbers examined their claim would seem to be justified. Their list comprises 494 Chinese foods, 167 Indian, 77 Malayan, and 32 Japanese foods, 770 in all. The results are presented in two large tables. In the first are given : the local name of the food, the ingredients of any made-up dishes, the methods of preparation (recipes, in fact), the nationality of those who prepare them (Tamils, Malayans, Brahmins, various Chinese district inhabitants, Javanese, Japanese, etc.), the method of eating (hot or cold, on a plate, in a bowl, with fork, spoon, chop-sticks or fingers), the average numbers of customers that the purveyor supplies with the particular food in question, the hours of sale, price, weight of a portion in grammes. In the second table are given : the results of chemical and biological analysis ; the percentages of water, mineral, protein, fat, carbohydrate, fibre, and calories ; the calories in a portion as purchased, and the cost in cents of 100 calories and of one gramme of protein. In a third table is shown the general distribution of the cost of energy ; half of the foods cost less than 1½ cents per 100 calories, and practically two-thirds (65 per cent.) yield this amount of energy for less than 2 cents. The cost of protein is consequently insignificant, but to meet physiological requirements careful choice of foods is necessary.

For those engaged in sedentary or non-manual work 2,400 calories and 1 gm. of protein per kgm. body weight, or about 56 gm. of protein, should be regarded as the basal requirement for adults, according to the League of Nations Health Committee.

Two things are needed to make the work complete ; one is a knowledge of the vitamin content of the foods. This, as the authors state, could not be done without adding several years of work to that already done and to wait for this would mean unreasonable delay. The title of the work, moreover, shows that there was no intention to include this aspect of food analysis and value. The second is, in the reviewer's opinion, a defect, which fortunately could be remedied without much trouble and should be remedied soon by the issue of a supplementary brochure giving, where possible, the scientific names as well as the local names of the foods used. If future issues of the publication are called for, this could be inserted in brackets in the same column as the local name or in the " ingredients " column, and so obviate any resetting of Table I. One or two examples may be given : Udang Goreng is the name given to a food containing prawns, flour and other ingredients, Udang Kring Goreng Suon to one with prawns, lobak and others. Unless we know that lobak is the local name for *Raphamus radicola*, we have to guess whether it is fish, fowl, animal, vegetable, or mineral. Kang Hoi is a preparation of crabs, Kang Huan Chui one of sweet potatoes ; Brinjal and Dhall curry conveys little to workers elsewhere than the east, but if we know that brinjal is *Solanum melongena* (the egg plant) we can apply the knowledge to varieties met with elsewhere ; as regards dhall, we know not which species is

meant, *Maeura dhall, Erbum Lens, Arar dhall, Cajanus indicus*, or *Mung dhall, Phaseolus Mungo*. Six varieties of kachang are mentioned, Kachang Kara, Kachang Kuda, Kachang Manis, Kachang Mera, Kachang Panjang, Kachang Parang, but no indication is given that kachangs do not all belong even to the same genus ; there is a kachang idjo (*Phaseolus radiatus*), Kachang Kedele (*Soja hispida*) the soy bean, Kachang panjang (*Vidrea catjang*), Kachang manis, the ground nut, *Archis hypogaea*. If the scientific names were given workers in other parts of the world would find these analyses of untold value and time spent in discovering whether a foodstuff mentioned is known locally to them under a different name would be saved.

It would not be fair to end a review of this work on a carping note. The task must have been a laborious one and has been brought to a most successful issue and the authors deserve the fullest praise for having accomplished so much. A limited number of copies are available and until the stock is exhausted those desiring to have a copy should apply to the Bureau of Hygiene and Tropical Diseases, which has been entrusted with their distribution. *H. H. S.*

TALICE (Rodolfo V.), COSTA (Radamés S.), RIAL (Benito) & OSIMANI (Juan J.). **Los 100 primeros casos agudos confirmados de enfermedad de Chagas (Tripanosomiasis americana) en el Uruguay. Estudio epidemiológico, clinico y parasitológico.**—349 pp. With 2 coloured plates & 162 figures. 1940. Montevideo : Libreros—Editores A. Monteverde y Cia. " Palacio del Libro."

This work probably contains all that is known and all that can be told concerning Chagas's disease in Uruguay at the present time. The information given is based, as regards the history, epidemiology and transmission, mostly on the published literature, and, as regards the clinical features, on personal study of 101 cases, which are detailed in an addendum.

Concerning the first part there is little to say beyond attesting to the excellent summary given by the authors. The information, being gathered from the literature, is already known to readers of this *Bulletin*, but it is very useful to have the evidence thus brought together in one work, and much delving for separate abstracts over a period of years will be saved. Under epidemiology are included the various insect vectors and their habitats, their geographical distribution in Uruguay, the animal hosts found infected in nature and their localization ; the morbidity and mortality among the inhabitants. Under the clinical section the symptomatology is discussed, stress being laid upon the early local oedema of the eyelids (Romaña's sign), and a score of good illustrations of this are produced ; others follow showing the dacryoadenitis. Next dealt with are special symptoms such as the fever, adenopathy, hepatomegaly, cutaneous manifestations and circulatory disturbances, each with reference to illustrative cases. A point on which readers will look for enlightenment is the goitre question : whether the authors conclude, with Chagas, that there are goitrous or myxoedematous forms of the disease, or whether (with most modern authors) the two conditions are accidental concomitants—Chagas's disease in the inhabitants of a goitrous district. Only two cases of thyroid involvement are mentioned in the list of cases. One was a boy of 10 years in whom the enlargement was slight (a photograph is given), the other a girl of 11–12

years in whom the enlargement was even less and for this the explanation is offered that the condition may have been physiological, associated with the onset of puberty. In view of this it seems hardly necessary to postulate an "endocrinotropic strain of *Trypanosoma cruzi*"; on the contrary, on such flimsy evidence as this, it can tend only to complicate the problem.

A section is devoted to diagnosis, divided into clinical and laboratory methods; the former based on symptoms, the latter on examination of blood-smears, on the results of animal inoculation, on xenodiagnostic methods, and the Machado-Guerreiro complement fixation test. The work is authenticated by a fairly full bibliography of local (Uruguayan) and of foreign (non-Uruguayan) references. Photographs of patients and photomicrographs of tissue changes are well reproduced and there are two fine coloured plates, one depicting *Triatoma rubrovaria* and *T. infestans*, the other several forms of *Trypanosoma cruzi* as seen in human blood. *H. H. S.*

ARMY MEDICAL LIBRARY, WASHINGTON: **Medicofilm Service of the Army Medical Library, Washington, its Purpose and Plan of Operation, together with a List of More than 4,000 Abbreviated Titles of Medical Periodicals currently received by this Library, September, 1940.**—26 pp. Washington: 7th St. and Independence Avenue.

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Am. signifies Amoebiasis and Amoebic Dysentery.
Bb. ,, Beriberi.
Bl. ,, Blackwater.
B.R. ,, Book Review.
Chl. ,, Cholera.
Der. ,, Tropical Dermatology.
Diet. ,, Deficiency Diseases including Epidemic Dropsy.
Dys. ,, Dysentery (Bacillary and Unclassed).
Fev. ,, Fevers.
Hel. ,, Helminthiasis.
Leish. ,, Leishmaniasis.
Lep. ,, Leprosy.
Lept. signifies Leptospirosis.
Mal. ,, Malaria.
Misc. ,, Miscellaneous.
Oph. ,, Tropical Ophthalmology.
Pel. ,, Pellagra.
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——, with Bose & Mukerji, 456 (Mal.)
—— & Chowhan, J. S., 466, 515 (Vms.)
——, Das Gupta, B. M., Sen, B. & Ahmed, Z., 377 (Dys.)
——, ——, —— & Hayter, R. T. M., 186 (Mal.)
——, Hayter, R. T. M. & Sen, B., 503 (Mal.)
——, Mazumdar, D. C. & Roy, A. C., 750 (Diet.)
——, Pasricha, C. L. & Banerjee, K., 751 (Diet.)
——, —— & Lal, S., 588 (Hel.)
—— & Rao, S. S., 656 (Hel.)
——, Roy, D. N., Hayter, R. T. M. & Sen, B., 746 (Mal.)
Chorine, V., 50 (Lep.)
——, with Marchoux, 49 *bis* (Lep.)
Chortis, P., 459 (Mal.)
Chow, C. Y., with Hsu, 151 (Hel.)
Chowhan, J. S., with Chopra, 466, 515 (Vms.)
Christophers, R., 379 (Misc.), 848 (Fev.)
Christophers, S. R., 174 (Mal.)
—— & Fulton, J. D., 190, 745 (Mal.)
Chu, F. T., with Yuan & Lee, 346 (Leish.)
Chu, I., Chung, Liu & Wang, 789 (Mal.)
Chub, G. M. & Panferova, E. A., 130 (Mal.)

Chung, H.-L., Khoo, F.-Y., Chang, S. K. P. & Ma, W. S., 119 (Am.)
——, Liu, Wang, C. W. & Chu, I., 789 (Mal.)
——, with ——, 261 *bis* (Fev.)
—— & Wang, C. W., 769 (Leish.)
——, with Wang, 769 (Leish.)
Ciavaldini, 837 (Bl.)
di Cio, A. V., with Castex, 123 (Am.)
Ciuca, M., with Ionesco-Mohaesti, Balteanu & Combiesco, 255 (Fev.)
Clapham, P. A., (214) (Hel.)
Clark, H. C., Komp, W. H. W. & Jobbins, D. M., 668 (Mal.)
Claudian, I., with Grecu, Ionescu & Constantinescu, 441 (Pel.)
——, with Ionescu & Constantinescu, 446 (Pel.)
Clausen, N. M., with Longley & Tatum, 12 (Tryp.)
Cleave, T. L., 257 (Fev.)
Cleckley, H. M., Sydenstricker, V. P. & Geeslin, L. E., 442 (Pel.)
Clemens, H. H. & Barnes, M. L., 817 (Misc.)
Cleveland, D. E. H. & Turvey, S. E. C., 186 (Mal.)
Cluver, F. W. P., 866 (Mal.)
Coatney, G. R., with Young & Stubbs, 783 (Mal.)
Cochrane, R. G., (330), 635 (Lep.)
——, Pandit, C. G. & Menon, K. P., 331 (Lep.)
——, Raj, M. P. & Roy, E., 342 (Lep.)
Coggeshall, L. T., 670 *bis* (Mal.)
——, with Eaton, 368 *bis* (Mal.)
Cohen, W., 674 (Misc.)
Colas-Belcour, J. & Romaña, C. (347), (Leish.)
Collier, D. R., with Oberdorffer, (42), 47 (Lep.)
Collier, W. A., with Mochtar, 644, (645) (Lept.)
Collignon, E., (507) (Mal.)
Colonial Development Fund Scheme, 695 (Tryp.)
Combiesco, D., with Ionesco-Mihaesti, Ciuca & Balteanu, 255 (Fev.)
Concepcion, I. & Camara, S. F., with Fulgencio, B., 630 (Lep.)
—— & Paulino, P., with Camara, S. F., & Gargaritano, M. L., 755 (Bb.)
Congo Belge, 397, 398 (Tryp.), 419 (Pl.), 522 (Misc.)
Connell, F. H. & French, H. T., 111 (Am.)
Connell, W. K., 813 (Misc.)
Constantinescu, P., with Grecu, Ionescu & Claudian, 441 (Pel.)
——, with Ionescu & Claudian, 446 (Pel.)
Coon, A. B., 406 (Tryp.)
Coppola, M. A., 601 (Mal.)
Cordoba, 237 (B.R.)
Cornejo, A., with Mazza, 229 (Misc.)
Corradetti, A., 358 *bis*, 452, 665, 743, (743), 793 *ter* (Mal.)
Corson, J. F., 9, 10 (Tryp.)
Costa, A., 588 (Hel.)
da Costa, A. M., 194 (Rab.)
Costa, H. M., with de Almeida, (46) (Lep.)
Costa, R. S. & Salveraglio, F. J., 516 (Vms.)
——, with Talice & Miranda, (145) Tryp.)
——, with —— & Osimani, 145 (Tryp.)
——, with —— Rial & Osimani, 889 (B.R.)
Coujard, R., with Champy, 753 (Bb.)
Courtice, R., with Lee, 879 (Misc.)
Cova-Garcia, P., (500) (Mal.)
Covell, G., (188), 598, 866 (Mal.)
—— & Afridi, M. K., 747 (Mal.)
—— & Harbhagwan, 740 (Mal.)
Cowdry, E. V., Ravold, A. & Packer, D. M., 50 (Lep.)
Cowen, D., with Wolf & Paige, 236 (Misc.)
Cox, G. W., with Parker, Kohls & Davis, 254 (Fev.)
Cox, H. R., 254, 268, 852 (Fev.)
—— & Bell, E. J., 562, 580 (Fev.)
Craciun, E., with Danielopolu, 566 (Fev.)
——, with ——, Lupo & Petresco, 567 *bis* (Fev.)
Craig, C. F., 791 (Mal.)
Craig, G. M., 115 (Am.)
Cram, E. B. & Folan, J. P., 480 (Hel.)
—— & Nolan, M. O., 300 (Hel.)
Crook, R. L., 177 (Mal.)
Cros, R., 496 (Mal.)
Crowell, R. L., 861 (Mal.)
Crown Agents for the Colonies, 819 (B.R.)
Crozafon, C., 402 (Tryp.)
Cruveilhier, L., 196 (Rab.)
Cuénod, with Burnet & Nataf, 448 (Oph.)
——, with ——, —— & Roussel, 873 (Oph.)
Culbertson, J. T., 409 (Tryp.)
—— & Wotton, R. M., 407 (Tryp.)
Curatolo, A., with de Muro, 871 (Dys.)

D.

Daengsvang, S., 307 (Hel.)
Dale, H., 700 (Tryp.)
Damboviceanu, A. & Barber, C., 721 (Chl.)
Dang-Van-Ngu, 96 (Der.)
Danielopolu, D. & Craciun, E., 566 (Fev.)
——, Lupo, M., Craciun, E. & Petresco, M., 567 *bis* (Fev.)
Danilova, M. I. & Buduimko, F. A., 505 (Mal.)
Danzel, L., 427 (Pl.)
Dartevelde, E., 150 (Hel.)
Das, B. B., (723) (Chl.)
Das, N., 636 (Lep.)
Das, P. C., with Lahiri & Malik, 718 (Chl.)
——, with Pasricha, Lahiri & Paul, 276 (Chl.)
Das Gupta, B. M., 184 (Mal.)
——, with Chopra, Sen & Ahmed, 377 (Dys.)
——, with ——, —— & Hayter, 186 (Mal.)
Dassanayake, W. L. P., 302 (Hel.)
Davey, J. B., 7 (Tryp.)
Davey, T. F., 48, 624 (Lep.)
David, 22 (Y. & S.)
Davis, G. E., 202 *bis*, 639 (R.F.), 562 (Fev.)
——, with Parker, Kohls & Cox, 254 (Fev.)
——, with Philip, 640 (R.F.)
—— & Walker, M. E., 640 (R.F.)
Davis, J. P., (441) (Pel.)
Davis, L. J., 207 (Lept.)
Dawson, J. R., Jr., 191 (Rab.)
Dawson, W. T., with Sanders, 185 (Mal.)
De, M. N. & Tribedi, B. P., 232 (Misc.)
——, with ——, 749 (Diet.)

De, N. K., with Dharmendra, (337) (Lep.)
De, R. K., 601 (Mal.)
De, S. S., 461, 512, 513 (Vms.)
Decourt, P., Belfort, J. & Schneider, J., 135 (Mal.)
——, Schneider, J. & Kerrest-Grosdidier, 458 (Mal.)
DeEds, F., with McNaught & Beard, 309 (Hel.)
Degotte, J., (43) (Lep.)
De la Barrera, J. M., 423 (Pl.)
Deland, C. M., 265 (Fev.)
Delanoe, E., 44, (635) (Lep.)
De la Paz, G. C., (235) (Misc.)
De la Rivière, R. D., 570 (Fev.)
Delbove, P., 265 (Fev.)
—— & Reynes, V., 573 (Fev.)
Delinotte, H., 636 (Lep.)
Delperdange, G., with Jadin, 480 (Hel.)
Del Ponte, E., (789), 860 (Mal.)
Del Río, J., with Earle, Pérez & Arzola, 178 (Mal.)
Del Vecchio, G., 743 (Mal.)
Dempster, G., 22 (Y. & S.)
Denecke, K., 632 (Lep.)
—— & von Haller, E., 140 (Tryp.)
Denison, G. A. & Dowling, J. D., 196, 621 (Rab.)
—— & Leach, C. N., 621 (Rab.)
Dennie, C. C., with Gilkey, 206 (R.B.F.)
Dennis, E. W., with Lund, 531 (Misc.)
Dérobert, L., 158 (B.R.)
Derrick, E. H., with Burnet, Freeman & Smith, 253 (Fev.)
—— & Smith, D. J. W., 851 (Fev.)
Descazeaux, J. (217) (Hel.)
——, with Roubaud, (217) (Hel.)
Deschiens, R., 217 (Hel.), 727 (Am.)
Devignat, R., 828 (Pl.)
——, with Lewillon & Schoetter, 828 (Pl.)
Devine, J., 700 (Tryp.)
Dey, A. C., 813 (Misc.)
Dey, N. C., with Maplestone, 97, 98 (Der.)
Dhar, D. R., 348 (Leish.)
Dharmendra, (328), 337 (Lep.)
—— & Chatterji, S. N., 343, 632 (Lep.)
—— & De, N. K., (337) (Lep.)
—— & Mukherji, N. N., 628 (Lep.)
Diamanti, G., 504 (Mal.), 524 (Misc.)
Dias, E., (417), 712 (Tryp.)
——, with Travassos, 574 (Fev.)
Digonnet, L., with Lwoff, Querido & Garnier, 435 (Pel.)
Dikshit, 292 (Mal.)
Dikshit, B. B., 745 (Mal.)
——, with Sokhey, 828 (Pl.)
Dinger, J. E., (95) (Y.F.)
Dinnik, J. A. & Dinnik, N. N., 310 (Hel.)
Dinnik, N. N., 310 (Hel.)
Discombe, G., with Vartan, 865 (Mal.)
Djoehana, R. M., with Assien Tahar, Eerkens & Sardjito, 768 (Leish.)
Do Amaral, A. D. F., with Galvão, 597 (Hel.)
Dobladez, M. J. L., with Mazza & Giordano, 714 (Tryp.)
Dobler, M., with Hegner, 508 (Mal.)
Dollfus, R., 214 (Hel.)
Dolmatova, A. V., with Shchurenkova, 381 (Misc.)
Dominguez, F., 679 (B.R.)
Doull, J. A. & Megrail, E., 627 (Lep.)
Do-van-Hoanh, with Montel, 599 (Mal.)
——, with ——, Le-Van-Phung & Tran-Van-Hanh, 43 (Lep.)
Dove, W. E., with Hull & Platts, 235 (Misc.)
Dowling, J. D., with Denison, 196, 621 (Rab.)
Drummond, J., 868 (Dys.)
Drummond, M., 228 (Misc.)
Dubois, A., 158 (B.R.), 642 (R.B.F.)
——, with Gavrilov & Fester, Mme., 333 (Lep.)
—— & Kohn, I., 410, 701 (Tryp.)
—— & Losner, S., 411 (Tryp.)
—— & Pearson, Y., 199 (R.F.), 206 (R.B.F.)
—— & Radna, R., (43) (Lep.)
—— & Resseler, R., 340 (Lep.)
—— & Vitale, S., 306 (Hel.)
Dubovskoj, P. A., 351 (Leish.)
Duggan, J. N. & Chitnis, V. K., 874 (Oph.)
Duke, H. L., 11 (Tryp.)
Dumon, G., with Mattei, Giraud & Dumon-Lenègre, Mme., 349 (Leish.)
Dumon-Lenègre, Mme., with Mattei, Giraud & Dumon, 349 (Leish.)
Duncan, D., 451 (Mal.)
Duncan, J. T., Martin, P. H. & Murgatroyd, F., 96 (Der.)
Dunn, M., with Boyd, 189 (Mal.)
Dupoux, R., Barthas, R., Antoine, A. & Garali, M. T., 134 (Mal.)
Durand, P. & Giroud, P., 579, 849, 850 (Fev.)
——, —— & Sparrow, H., 849 (Fev.)
—— & Sparrow, H., 572, 849 (Fev.)
Durand, R., with Laigret, 266, 267, 569 (Fev.)
Duren, A., (287) (Mal.)
Durieux, C., with Peltier, Jonchère & Arquié, 92, 557 (Y.F.)
Dutta, S. C., with Hare, 69 (Hel.)
Duvoir, M., Pollet, L., Brumpt, L. C. & Chénebault, J., 596 (Hel.)
Dyer, R. E., 571 (Fev.)
——, with Lillie & Topping, 572 (Fev.)
——, with Topping, 843 (Fev.)
Dyke, H. W., 329 (Lep.)

E.

Earle, H. G., 757 (B.R.)
Earle, K. V., 225, 812, 815 (Misc.)
Earle, W. C., 495 (Mal.)
——, with Boyd, 288 (Mal.)
——, Pérez, M., Del Rio, J. & Arzola, C., 178 (Mal.)
Eaton, M. D. & Coggeshall, L. T., 368 *bis* (Mal.)
Edward, D. G., with Nabarro, 362 (Mal.)
Edwards, F. W., Oldroyd, H. & Smart, J., 387 (B.R.)
Eerkens, J. W., with Assien Tahar, Djoehans, & Sardjito, 768 (Leish.)
Egypt, Ministry of Public Health, 450 (Oph.)
El Bakly, M. A., 871 (Oph.)
El Ghariny, A., with Alport & Ghalioungui, 437 (Pel.)
Eliche, J., with Farinaud, 185 *bis* (Mal.)
Ellis, F. A., 406 (Tryp.)

Elvehjem, C. A., (805) Pel.)
——, with Waisman, Mickelsen, & McKibbin, (797) (Pel.)
Enders, J. F., with Zinsser & Plotz, 580 (Fev.)
Ermakova, N., 629 (Lep.)
Etcheverry, 46 (Lep.)
Ethes, Y., with Sicé & Sautet, 857 (Mal.)
v. Euler, H. & Schlenk, F. (436) (Pel.)
Evans, E. F., 841 (Fev.)
Evans, W. S., 192 (Rab.)
Evers, L. B., 309 (Hel.)
Ewins, J. A., 700 (Tryp.)

F.

Fabiani, G., (38) (Lep.)
Fadel, A. E. L., 871 (Oph.)
Faiguenbaum, J., 504 (Mal.)
Failmezger, T. R. & Spalding, J. E., 308 (Hel.)
Fairley, N. H., 369, 833 (Bl.), 863 (Mal.), 885 (Misc.)
—— & Murgatroyd, F., 834 (Bl.)
Far Eastern Association of Tropical Medicine, 539 (B.R.)
Farinaud, E. & Eliche, J., 185 *bis* (Mal.)
——, Lataste, C., Baccialone, L. & Canet, J., 186 (Mal.)
—— & Prost, P., 289 (Mal.)
Farinaud, E. M., 128 (Mal.)
Farinaud, M. E., 136 (Mal.)
—— & Prost, P., 494 (Mal.)
Faust, E. C., 178, 356 (Mal.)
——, with Watson & Simmons, (53) (Mal.)
——, Sawitz, W., Tobie, J., Odom, V., Peres, C. & Lincicome, D. R., 62 (Hel.)
Favorite, G. O., 531 (Misc.)
Federated Malay States, 131, 284, (Mal.), 620 (Rab.), 831 (Pl.), 847 (Fev.)
Fein, H. D., with Jolliffe & Rosenblum, 438 (Pel.)
Feissolle, L., with Raynal & Lieou, 720 (Chl.)
Fenner, F. J., 23 (Y. & S.)
Fernandez, J. M, M., 632 (Lep.)
Fernández-Lascano, F., with Talice, Mourigán & Osimani, 145 (Tryp.)
Ferwerda, S., with Tenhaeff, 591 (Hel.)
Fester, A., Mme., with Gavrilov, 333 (Lep.)
—— with —— & Dubois, 333 (Lep.)
Fici, V., 227 (Misc.)
Fiebiger, W. & Tempel, D. (211) (Hel.)
Field, H., Jr. & Robinson, W. D., 440 (Pel.)
Field, J. W., 808 ,874 (Misc.)
—— & Le Fleming, H., 56, 739 (Mal.)
Findlay, G. M., 848 (Fev.)
—— & MacCallum, F. O., 93 *bis* (Y.F.)
——, with Murgatroyd & MacCallum, 129 (Mal.)
Finkelstein, B. J., 621 (Rab.)
Finlayson, M. H., 518 (Vms.)
—— & Grobler, J. M., 581 *bis* (Fev.)
Fischer, I. A., 677 (Misc.)
Flarer, F., 776 (Leish.)
Floch, H., Goerger, F. & Tasque, P., 645 (Lept.)
Floresco, P., with Gingold, 130 (Mal.)
Florman, A. L. & Hafkenschiel, J., 845 (Fev.)
Folan, J. P., (300) (Hel.)
——, with Cram, 480 (Hel.)
Fondé, G. H. & Fondé, E. C., 502 (Mal.)
Foner, A., 532 (Misc.)
Foraco, J., 193 (Rab.)
Forster, W. G., 872 (Oph.)
Fournials, 28 (Sp.)
Fournier, J., with Raynal, 256 (Fev.)
——, with —— & Velliot, 256 (Fev.)
Fox, H., 105 (Der.)
Fratani, L., 496 (Mal.)
Freeman, M., with Burnet, 563 (Fev.)
——, with ——, Derrick & Smith, 253 (Fev.)
Freire, F., with Vargas, 784 (Mal.)
French, H. T., with Connell, 111 (Am.)
Frimodt-Möller, C., 387 (Misc.)
Frohne, W. C., 181 (Mal.)
Frostad, S., 35 (Sp.)
Frostig, J. P., with Lewy, Himwich & Spies, (444) (Pel.)
—— & Spies, T. D., 443 (Pel.)
Frye, W. W. & Meleney, H. E., 115 (Am.)
——, with —— & Leathers, 115 (Am.)
Fuchs, H. & Wisselinck, A., 33 (Sp.)
Fuente-Méndez, R., with Talice, (145) (Tryp.)
Fuertes, F. & Peruchena, J. G., 334 (Lep.)
Fulgencio, B., with Camara & Concepcion, 630 (Lep.)
Fulton, J. D., 509, 795 (Mal.)
——, with Christophers, 190, 745 (Mal.)

G.

Gabriel, W. B., 121 (Am.)
Gaceta Medica de Caracas, 231 (Misc.)
Galizzi, J., 870 (Dys.)
Gallardo, V. P., 714 (Tryp.)
Galliard, H., 152, 489, 650 (Hel.)
—— & Nguyen Huu Phiem, 305 (Hel.)
Galvão, A. L. & Do Amaral, A. D. F., 597 (Hel.)
Garali, M. T., with Dupoux, Barthas & Antoine, 134 (Mal.)
Garcia-Capurro, F., with Piaggio-Blanco, 589 (Hel.)
Gargaritano, M. L., with Concepcion, Paulino & Camara, 755 (Bb.)
Garnier, Mlle., with Lwoff, Querido & Digonnet, 435 (Pel.)
Garra, G., 865 (Mal.)
Gaschen, H., with Marneffe & Nguyen-Ba-Tung, 355 (Mal.)
Gasic, G., 414 (417) (Tryp.)
Gatto, I., 347 (Leish.)
Gaud, M. & Pocoule, A., 565 (Fev.)
Gautrelet, J., 463 (Vms.)
Gavrilov, W., Dubois, A. & Fester, Mme., 333 (Lep.)
—— & Fester, A., Mme., 333 (Lep.)
—— & Nijs, J., 808 (Misc.)
Gear, H. S., 156 (B.R.)
Gear, J., with de Meillon, 128, 739 (Mal.) 260, 263 (Fev.)
Gebert, S., 498 (Mal.)
Geens, J., with Van den Branden, 237 (Misc.)

Geeslin, L. E., with Checkley & Sydenstricker, 442 (Pel.)
——, with Sydenstricker, Schmidt & Weaver, 437 (Pel.)
——, with ——, Templeton & Weaver, 439 (Pel.)
Gehr, E., 626 *bis* (Lep.)
Geller, E. R., 742 (Mal.)
Genevray, J., 717 *bis*, 718 (Chl.)
——, Toumanoff, C. & Try, H. T., 293, 664 (Mal.)
Genofre, W., with Pinotti, Romeu & Viana, 824 (Pl.)
George, J. V., 330 (Lep.)
Georgiadès, D., 402 (Tryp.)
Gergawy, I. F., 273 (Fev.)
Germond, R. C., 338, 635 (Lep.)
Ghaliougui, P., with Alport, 733 (Am.)
——, with —— & El Ghariny, 437 (Pel.)
Ghidini, G. M., 401 (Tryp.)
Ghizzetti, C., 184 (Mal.)
Ghosh, H., 124 (Am.)
Giacobbe, M., 458 (Mal.)
Giglioli, G., 356 *quat.* (Mal.)
Gil de Castro Cerqueira, with Mendes, 336 (Lep.)
Gilkey, H. M. & Dennie, C. C., 206 (R.B.F.)
Gill, D. G., with Smith & McAlpine, 478 (Hel.)
Gillanders, G., 526 (Misc.)
Gilroy, A., 52 (Mal.)
Gil Yepez, C. (39) (Lep.)
Gimenez, C., with Alberto Aguirre, 415 (Tryp.)
Gingold, N. & Floresco, P., 130 (Mal.)
Giomo, G., 223 (Misc.)
Giordano, F., 863 (Mal.)
Giordano, J. J., with Mazza & Dobladez, 714 (Tryp.)
Giordano, M., 154 (B.R.)
Giovannola, A., 150, 151, 479, 485 (Hel.), 367 (Mal.)
Girard, G., 422, 829 (Pl.)
—— & Milliau, M., 620 (Rab.)
—— & Radaody-Ralarosy, P., 830 (Pl.)
Giraud, P., with Mattei, Dumon & Dumon-Lenègre, Mme., 349 (Leish.)
Girgis, S., 120 (Am.)
Giroud, P., 252 (Fev.)
——, with Durand, 579, 849, 850 (Fev.)
——, with —— & Sparrow, 849 (Fev.)
Gispen, R., 280 (Chl.)
—— with Loopuyt, 207 (R.B.F.)
Githens, T. S. & Wolff, N. O'C., 467 (Vms.)
Giunta, G. & d'Ignazio, C., 257 (Fev.)
Glass, S., with Rosenfeld, 511 (Vms.)
Gminder, E., 333 (Lep.)
Goerger, F., with Floch & Tasque, 645 (Lept.)
Gohar, N., 99 (Der.)
Gokhale, S. K., with Sokhey & Chitre, 427 (Pl.)
Gold Coast, 549 (Y.F.)
Goldfarb, A. I., with Herman, 508 (Mal.)
Goldsmith, G. A., Ogaard, A. T. & Gowe, D. F., 806 (Pel.)
Goldstein, F., with Manwell, 60, 508, 794 (Mal.)
de Golovine, S., 634 (Lep.)
Gomes, L. de S., 778 (Leish.)
González Barreras, P., 362 (Mal.)
González Rincones, R., 231 (Misc.)
Goodall, J. W. D., 804 (Pel.)
Gordon, H. M., 7 (Tryp.)
Gordon. R. M., (591) (Hel.)
——, with Bertam, 497 (Mal.)
—— & Lumsden, W. H. R., 653 (Hel.)
Gore, 28 (Sp.)
Gorlitzer, V., 868 (Dys.)
Gotten, H. B. & MacGowan, J. J., 836 (Bl.)
Gouget, R., (669) (Mal.)
Gowe, D. F., with Goldsmith & Ogaard, 806 (Pel.)
de Graaf, W., with Wolff, 262, 264 (Fev.)
Gradle, H., 447 (Oph.)
Grasset, E. & Schaafsma, A., 513 (Vms.)
Grecu, A., Ionescu, N. G., Claudian, I. & Constantinescu, P., 441 (Pel.)
Greenbaum, R. S., with Leob, 314 (Bb.)
Greene, R. A. & Breazeale, E. L., 194 (Rab.)
Greenfield, J. G. & Holmes, J. M., 430 (Pel.)
Greig, E. D. W., 658 (Hel.), 732 (733) *bis* (Am.)
—— & Neill, A., 361, 456 (Mal.)
——, with —— (601) Mal.)
Grenierboley, J., with Catanei, 97 (Der.)
Grenoilleau, J., with Lartigue, Megnin & Laurens, (366) (Mal.)
Greval, S. D. S., with Lowe, (43) (Lep.)
Grimes, C. & Lavergne, J., 864 (Mal.)
Grimes, M., 46 (Lep.)
Grobler, J. M., with Finlayson, 581 *bis* (Fev.)
Grott, J. W., 376, 379 (Dys.)
Grüneis, P. & Pilgerstorfer, E., 869 (Dys.)
Guiang, R. V., 314 (Bb).
Guigon, G., 351 (Leish.)
Guily, P., with Marill & Kessis, 291 (Mal.)
Guisoni, F., with Bernard, Alcay & Manya (150) (Hel.)
Gunasekara, S. T., 495 (Mal.)

H.

Hackett, L. W., with Bates, (856) (Mal.)
Haddad, E., 594 (Hel.)
Haex, A. J. C., with Kastein, 208 (Lept.)
Hafkenschiel, J., with Florman, 845 (Fev.)
Hakansson, E. G., 728 (Am.)
Halawani, A. & Sobky, M. F., 864 (Mal.)
Halder, K. C., with Smith & Ahmed, 769 (Leish.)
Hallay, L. T., 447 (Oph.)
von Haller, E., with Denecke, 140 (Tryp.)
Hamburger, H. J., 223, 812 (Misc.)
Hampton, B. C. 822 (Pl.)
Harán, A. M., 730 (Am.)
Harbhagwan, with Covell, 740 (Mal.)
Harding, R. D., 697 (Tryp.)
Hare, K. P., 529 (Misc.), 596 (Hel.)
—— & Dutta, S. C., 69 (Hel.)
Harwant Singh, with Afridi & Jaswant Singh, 500 (Mal.)
Hasselmann, C. M., 24 (Y. & S.)
Hasselmann-Kahlert, M., 878 (Misc.)
Haulussy, M. & Sardjito, 383 (Misc.)
Hausser, G., 598 (Mal.)
Hawaii, Territory of, 830 (Pl.)
Hawking, F., 306 (306) (Hel.), 697, 700, 701 (Tryp.)

Hayter, R. T. M., with Chopra, Das Gupta & Sen, 186 (Mal.)
——, with ——, Roy & Sen, 746 (Mal.)
——, with —— & Sen, 503, 746 (Mal.)
Headlee T. J., with Powers, 94 (Y.F.)
Health, Canberra, (559) (Y.F.)
Hees, E., 236 (Misc.)
Hegner, R. & Dobler, M., 508 (Mal.)
Henrard, C., with van Hoof & Peel, 703 *quat.* (Tryp.)
Henry Lester Institute of Medical Research, Shanghai, 757 (B.R.)
Herman, C. M. & Goldfarb, A. I., 508 (Mal.)
Herms, W. B., 79 (B.R.)
Hernan Mendez, 856 (Mal.)
Herr, A. & Brumpt, L., 146 (Tryp.)
Hertig, M., 853 (Fev.)
Hervel, J. E., with Webb, 739 (Mal.)
Heydon, G. A. M. & Bearup, A. J., 220 (Hel.)
Higgins, A. R., 282 (Chl.)
Hilgermann, R., 569 (Fev.)
Hill, R. B., with Cambournac, 784 (Mal.)
Hilmy, F., with Wakil, 273 (Fev.)
Himwich, H. E., with Lewy, Frostig & Spies, (444) (Pel.)
Hinman, E. H., (289) (Mal.)
—— & Hurlbut, H. S., 788 *bis* (Mal.)
Hoang-Tich-Try, with Toumanoff, 187 (Mal.)
Hoare, C. A., 710 (Tryp.)
Hodgkin, E. P., 302 (Hel.)
Hoff, H. & Shaby, J. A., 147, 592 (Hel.)
Hoffman, W. A. & Janer, J. L., 67 (Hel.)
Hoffmann, W. H. (95), 556 (Y.F.), 414 (Tryp.)
Holland, E., with de Meillon, 480 (Hel.)
Holland, G. P., 843 (Fev.)
Holmes, J. M., 35 (Sp.)
——, with Greenfield, 430 (Pel.)
Holmes, W. E., 136, 866 (Mal.)
Hong Kong, 818 (Misc.)
van Hoof, F., Henrard, C. & Peel, E., 703 *quat.* (Tryp.)
van Hoof, L., 397 (Tryp.), 419 (Pl.)
van Hoof, T., with Rodhain, 795 (Mal.)
Hope, R. B., with Ruddock, 534 (Misc.)
Hopkins, R., 626 (Lep.)
Horgan, E. S. & Kirk, R., 774 (Leish.)
Höring, F. O., 94, 95 (Y.F.)
Horner, I., 138 (Tryp.)
Horrenberger, R., with Béguet, 621 (Rab.)
Hou, H. C., 441 (Pel.), 635 (Lep.)
Howard, S. C., 866 (Mal.)
Hoyt, A. & Warner, D., 615 (Rab.)
Hsü, H. F., 152, 489 (Hel.)
—— & Chow, C. Y., 151 (Hel.)
Hu, S. M. K., 181 (Mal.)
Huang, C. H., 773 (Leish.)
Huard, P., 119, 733 (Am.)
—— & Bertrand, C., 225 (Misc.)
Hudson, H. W., Jr., with Botsford & Chamberlain, 648 (Hel.)
Hughes, W., 224 (Misc.)
Hull, J. B., Dove, W. E. & Platts, N. G., 235 (Misc.)
—— & Shields, S. E., 380 (Misc.)
Humphries, S. V., 523 (Misc.)
Hurlbut, H. S., with Hinman, 788 *bis* (Mal.)
Hutton, E. L. & Shute, P. G., 456 (Mal.)
——, with Sinton & Shute, 54, 55, 183 (Mal.)
Huzii, T., with Sakanaka, 372 (Dys.)
Hwang, M. S., with Morris & Kuo, 799 (Pel.)

I.

Ide, M., 723 (Chl.)
d'Ignazio, C., with Giunta, 257 (Fev.)
——, Lombardi, A. & d'Arcangelo, D., 565 (Fev.)
Ignacio, J. L., 335 (Lep.)
Ignacio Chala, J., (334) (Lep.)
Imbasciati, B., 95 (Y.F.)
Impallomeni, R., with de Muro, 375 (Dys.)
Inbona, J. M., with Justin-Besançon & Caroli, 32, 33 (Sp.)
Indian Medical Gazette, 232 (Misc.), 748 (Diet.), 769 (Leish.)
International Journal of Leprosy, 343 (Lep.)
Ionescu, N. G., Claudian, I. & Constantinescu, P., 446 (Pel.)
——, with Grecu, Claudian & Constantinescu, 441 (Pel.)
Ionesco-Mihaesti, C., Ciuca, M., Balteanu, I. & Combiesco, D., 255 (Fev.)
Iriarte, D. R., with Briceño Rossi, 105 *bis* (Tryp.)
Irisawa, T., (232) (Misc.)
Irvine, C., 342 (Lep.)
Irving, H., with Reid & Scherer, 817 (Misc.)
Iswariah, V. & Nair, V. G., 26 (Y. & S.)
Iturraspe, M. C., 588 (Hel.)
Ivanissevich, O., 587 (Hel.)
Iyengar, M. O. T., 652 (Hel.)
Izquierdo, F., 231 (Misc.)

J.

Jack, R. W., 400 (Tryp.)
——, 699 (Tryp.)
Jacob, V. P., with Russell, 506 *ter* (Mal.)
——, with ——, 53 (Mal.)
Jacobi, L., 59 (Mal.)
Jadfard, H., with Burnet, 331 (Lep.)
Jadin, J., 615 (Rab.), 734 (Am.)
—— & Arnaldi, E., 557 (Y.F.)
—— & Delperdange, G., 480 (Hel.)
Jakmauh, P. J., with Welch, 575 (Fev.)
Jakusheva, A. I., 451 (Mal.)
James, C. S., 185 (Mal.)
James, S. P., 59, 174 (Mal.), 88 (Y.S.)
Janer, J. L., 729 (Am.)
——, with Hoffman, 67 (Hel.)
Jaramillo, R., 272 (Fev.)
Jaswant Singh, with Afridi & Harwant Singh, 500 (Mal.)
Jaulmes, C., with Sohier, 114 (Am.)
Jerace, F., 285 (Mal.)
Joannon Infante, J., 258 (Fev.)
Jobbins, D. M., with Clark & Komp, 668 (Mal.)
Joelsas, M., 600 (Mal.)
Johansen, F. A., 343 (Lep.)
Johnson, C. G., 886 (Misc.)
Johnson, C. M., with Sapero, 111 (Am.)
Johnson, H. N., with Leach, 616 (Rab.)
Johnson, W. B., 329 (Lep.)

Jolliffe, N., Fein, H. D. & Rosenblum, L. A., 438 (Pel.)
Jolly, A. & Di Ruggiero, 266 (Fev.)
Jonchère, H., with Peltier, Durieux & Arquié, 92, 557 (Y.F.)
Jones, A. M. & Bramwell, C., 315 (Bb.)
Jones, F. R., with Ordman, 638 (R.F.)
Jonnesco, D., 192 (Rab.)
Joranson, Y. & Simons, J., 122 (Am.)
Jordan, J. H., 193 (Rab.)
——, Pedersen, H. & Shu, S., 192 (Rab.)
Jores, A. & Wolter, H., (70) (Hel.)
Jörg, M. E., 464 (Vms.)
——, with Mazza, 415 (Tryp.)
——, with —— Basso, G. & Basso, R., 414 (Tryp.)
——, with —— & Tobar, (714) (Tryp.)
Journal of the American Medical Association, 68, 309, 659 (Hel.), 196 (Rab.)
Joyeux, B., Truong-cam-Cong & Nguyen-xuan-Nguyen, 216 (Hel.)
Joyeux, C. & Sautet, J., 346 (Leish.)
——, Sicé, A. & Sautet, J., 286 (Mal.)
Julianelle, L. A., 872 (Oph.)
—— & Smith, J. E., 872 (Oph.)
Junge, W., 603 (Mal.), 676 (Misc.)
Junior, F. P., 832 (Pl.)
Junior, P., 501 (Mal.)
Jurchak, F. E. & Bozhenko, V. P., 295 (Mal.)
Juschko, S. S., 44 (Lep.)
Justin-Besançon, L., Caroli, J. & Inbona, J. M., 32, 33 (Sp.)

K.

Kachalova, E. K., with Serguiev, Nabokov & Zeifert, 506 (Mal.)
Kagy, E. S., 110 (Am.)
Kamitani, K., with Momma & Yamashita, 648 (Hel.)
Kark, R., with Meiklejohn, 434 (Pel.)
Kasahara, S., with Kawamura, Toyama, Nishinarite & Tsubaki, 269 (Fev.)
Kasauli, 598 (Mal.)
Kast, C. C., with Kolmer & Rule, 708 (Tryp.)
Kastein, G. W. & Haex, A. J. C., 208 (Lept.)
Kato, T., 488, 490 (Hel.)
Katzenellenbogen, I., 444 (Pel.)
Kauntze, W. H., 397 (Tryp.)
Kawabe, K., with Kobayasi, Yokoi & Tutumi, 856 (Mal.)
Kawamura, R., Kasahara, S., Toyama, T., Nishinarita, F. & Tsubaki, S., 269 (Fev.)
—— & Ueda, M., 269 (Fev.)
Kegel, R., with Yegian, 534 (Misc.)
Kellog, W. H., 622 (Rab.)
Kennedy, J. S., 553 (Y.F.)
Kerbosch, M., 602 (Mal.)
Kernevez, with Rivoalen & Bruneau, 578 (Fev.)
Kerrest-Grosdidier, with Decourt & Schneider, 458 (Mal.)
Kessis, R., with Marill & Guily, 291 (Mal.)
Kesteven, H. L., 97 (Der.)
Khartoum, 818 (Misc.)
Khoo, F.-Y., with Chung, Chang & Ma, 119 (Am.)
Khovanskaya, A. I., with Rashina, 133 (Mal.)
Kikuth, W. & Mudrow, L., 188, 671 *bis* (Mal.)
King, H., 700 (Tryp.)
King, W. V., (182) (Mal.)
Kingsbury, A. N., 131, 284 (Mal.)
Kinugasa, M., 490 (Hel.)
Kirk, R., 198 (R.F.)
—— with Aldridge, 871 (Oph.)
—— with Horgan, 774 (Leish.)
—— & Lewis, D. J., 770 (Leish.)
—— & Sati, M. H., 771 (Leish.)
Kirkland, W. B., (887) Misc.)
Kirwan, E. O'G., 449 (Oph.)
Kitchen, S. F., 179 (Mal.)
Kleeman, I., with Lichterman, 660 (Hel.)
Kleine, F. K. & Kunert, H., 702 (Tryp.)
Kligler, H., with Kligler & Olitzki, 710 (Tryp.)
Kligler, I. J., Olitzki, L. & Kligler, H., 710 (Tryp.)
Kmecza, J. M., 531 (Misc.)
Knipe, F. W., with Russell, 504 (Mal.)
Knott, J., 304, 655 (Hel.)
Ko, E., 153 (Hel.)
Kobayasi, H., 651 (Hel.)
——, with Yokogawa & Yosino, 655 (Hel.)
——, with —— Yumoto, Osaka, Ro & Yokogawa, M., 651 (Hel.)
——, Yojoi, K., Kawabe, K. & Tutumi, S., 856 (Mal.)
Kodicek, E., (797) *bis* (Pel.)
Kohls, G. M., with Parker, Cox & Davis, 254 (Fev.)
Kohn, I., with Dubois, 410, 701 (Tryp.)
Kolmer, J. A., Kast, C. C. & Rule, A. M., 708 (Tryp.)
Kolochine-Erber, B. & Stefanopoulo, G. J., 643 (Lept.)
Kolodny, M. H., 140, 143, 408 (Tryp.)
Komp, W. H. W., with Clark & Jobbins, 668 (Mal.)
Konakhov, A. G., with Aleksejev, 345 (Leish.)
Kondo, H., 116 (Am.)
Konj, J. S. & Poleshko, G. V., 506 (Mal.)
Kooser, J. H. & Blankenhorn, M. A., 440 (Pel.)
Kopp, I. & Solomon, H.C., 705 (Tryp.)
Kotter, G. F., 208 (Lept.) 577 (Fev.)
Kouwenaar, W., 846 (Fev.)
Kovacs, L., with Van den Berghe, 129 (Mal.)
Kraneveld, F. C. & Umboh, T. E. W., 412 (Tryp.)
Krause, M., with Mainzer, 487 (Hel.)
Krikorian, K. S. & Bedrechi, N., 682 (B.R.)
Krishnan, B. G., with Aykroyd & Passmore, 431 (Pel.)
Krishnan, K. V., 744, 858 (Mal.)
Kröber, F., 226 (Misc.)
von Krogh, M., 110 (Am.)
Kubo, M. & Ohito, E., 365 (Mal.)
Kuczynski-Godard, M. H., 564 (Fev.), 754 (Bb.)
Kudo, R. R., 756 (B.R.)
Kumm, H. W. & Ruiz, H., 357 (Mal.)
Kunert, H., 17 (Tryp.)
——, with Kleine, 702 (Tryp.)
Kuo, P. T., 312 (Bb.)
——, with Morris & Hwang, 799 (Pel.)
Kurotchkin, T. J., with Mu, 98 (Der.)

L.

Labernadie, V., (341), 634 (Lep.)
Laborde, A., 376 (Dys.)
La Face, L., 295 (Mal.)
Lagrosa, M., 335 (Lep.)
Lahiri, M. N., Das, P. C. & Malik, K. S., 718 (Chl.)
——, with Pasricha, Das & Paul, 276 (Chl.)
Laidlaw, P., 627 (Lep.)
Laigret, J., 850 (Fev.)
—— & Durand, R., 266, 267, 569 (Fev.)
Lal, R. B., 717 (Chl.)
Lal, S., with Chopra & Pasricha, 588 (Hel.)
——, with Pasricha & Banerjee, 749 (Diet)
——, with ——, Malik & Biswas, 750 (Diet.)
Lall, R. & Townsend, R. S., 384 (Misc.)
Lambert, S. M., 70 (Misc.)
Lamborn, W. A., 824 (Pl.)
Lamborn, W. A. S., 370 (Bl.)
Lambrechts, A., with Brull & Barac, 34 (Sp.)
Lamote, 126 (Mal.)
Lancet, 576 (Fev.)
Landsberg, J. W., with Otto, 593 (Hel.)
Lane, C., 477 (Hel.)
Langauer, L., 624 (Lep.)
Lara, C. B., 335, 632 (Lep.)
Larghero-Ybarz, P., 590 (Hel.)
Lartigue, J., Grenoilleau, J., Megnin, J. & Laurens, L., (366) (Mal.)
Lassman, P., 811 (Misc.)
——, with Rodhain, 459, 509, 794 (Mal.)
Lataste, C., with Farinaud, Baccialone & Canet, 186 (Mal.)
——, with Querido & Lwoff, 434 (Pel.)
Lattouf, A. G., 733 (Am.)
Laurens, L., with Lartigue, Grenoilleau & Megnin, (366) (Mal.)
Lavarello, A. O., (805) (Pel.)
Lavergne, J., with Grimes, 864 (Mal.)
Lavery, F. J., 447 (Oph.)
Lavier, G., 148 (Hel.), 869 (Dys.)
——, Bariéty, M., Caroli, J. & Boulanger, P., (153) (Hel.)
——, with Brumpt, 151 (Hel.)
—— & Leroux, R., 401 (Tryp.)
Lawkowicz, W., 271 (Fev.)
Lawler, H. J. (650) (Hel.)
Lawson, T. C., 212 (Hel.)
Leach, C. N., with Denison, 621 (Rab.)
—— & Johnson, H. N., 616 (Rab.)
League of Nations, Health Organisation, 757 (B.R.)
Leathers, W. S., with Meleney & Frye, 115 (Am.)
Le Cosquino de Bussy, I. J. & van Loghem, J. J., 266 (Fev.)
Lee, C. U., with Yuan & Chu, 346 (Leish.)
Lee, D. H. K. & Courtice, R., 879 (Misc.)
Leeson, H. S., 453 (666), (Mal.)
——, with de Meillon, (665) (Mal.)
Le Fleming, H., with Field, 56, 739 (Mal.)
Le Gac, P., 575 (Fev.)
——, Samara, M. & Servant, J., 275 (Fev.)
—— & Servant, J., 18 (Tryp.), 273 (Fev.)
Le Gaonach, J., 500 (Mal.)
Lehmann, J. & Nielsen, H. E., 313 (Bb.)
Leite, A. L., 416 (Tryp.)
Lent, H., 713 (Tryp.)
—— & Pifano, F., 142 (Tryp.)
Lenti, P. & Serra, G., 23 (Y. & S.)
Leonida, J., (807) (Pel.)
Lépine, P., 620 (Rab.)
——, Mathis, M. & Sautter, V., Mlle., 614 (Rab.)
Leprosy Review, (45), 328 (Lep.)
Lepsi, I., 494 (Mal.)
Le Renard, A. & Raynaud, M., 258 (Fev.)
Leriche, E., 790 (Mal.)
Leroux, R., with Lavier, 401 (Tryp.)
Lesné, Cayla & Lichtenberger, 362 (Mal.)
Lester, H. M. O., 7 (Tryp.)
Lê-Thi-Van, with Seyberlich, 814 (Misc.)
Le Van, J. H., 555 (Y.F.)
Le-Van-Phung, with Montel, Do-Van-Hoanh & Tran-Van-Hanh, 43 (Lep.)
Levine, N. D., 373 (Dys.)
Levitanskaia, P. B., 373 (Dys.)
Levy, A. H., 449 (Oph.)
Lewillon. R., Devignat, R. & Schoetter, M., 828 (Pl.)
Lewis, D. J., 454 (Mal.)
——, with Kirk, 770 (Leish.)
Lewis, J. T., 213 (Hel.)
Lewthwaite, R., 620 (Rab.), 642 (R.B.F.), 847 (Fev.)
—— & Savoor, S. R., 576, 847 (Fev.)
Lewy, F. H., Himwich, H. E., Frostig, J. P. & Spies, T. D. (444) (Pel.)
——, Spies, T. D. & Aring, C. D., 805 (Pel.)
Lichtenberger, with Lesné & Cayla, 362 (Mal.)
Lichterman, A. & Kleeman, I., 660 (Hel.)
Lieb, F., 719 (Chl.)
Lie Kian Joe, with Bonne, 220 (Hel.)
Lieou, Y. C., 614, 615 (Rab.)
——, with Raynal, 614, 621 (Rab.)
——, with —— & Feissolle, 720 (Chl.)
Lieu, V. T., with Shih & Wu, 122 (Am.)
Lillie, R. D., Dyer, R. E. & Topping, N. H., 572 (Fev.)
——, with Roe & Wilcox, 876 (Misc.)
Lima, J. P. C. & Arantes, M., 332 (Lep.)
Lima, L. de S., (39) (Lep.)
Lincicome, D. R., with Faust, Sawitz, Tobie, Odom & Peres, 62 (Hel.)
——, with Sawitz & Odom, 62 (Hel.)
Lindberg, K., 659 *bis* (Hel)
Lion, M., 367 (Mal.)
Lipscomb, F. M., (292) (Mal.)
Liu, C. Y., (425) (Pl.)
Liu, P. Y., with Zia & Pang, 579 (Fev.)
Liu, W. T. & Chung, H. L., 261 *bis* (Fev.)
——, with ——, Wang & Chu, 789 (Mal.)
Livadas, G., Canellakis, A. & Valaoras, V. G., 291 (Mal.)
Livesco, M., 442 (Pel.)
Lleras Restrepo, E., 344 (Lep.)
Lloyd, L., 379 (Misc.)
Lodenkamper, H., 717 (Chl.)
Loeb, L. M. & Greenbaum, R. S., 314 (Bb.)
Loewenthal, L. J. A., 100, 101, 102, 103, 104 (Der.)
van Loghem, J. J., with Le Cosquino de Bussy, 266 (Fev.)

Lombardi, A., with d'Ignazio & d'Arcangelo, 565 (Fev.)
Longley, B. J., Clausen, N. M. & Tatum, A. L., 12 (Tryp.)
van Lookeren Campagne, J., 219 (Hel.)
Loopuyt, L. & Gispen, R., 207 (R.B.F.)
Lopez-Gutierrez, J. C., with Prat, 590 (Hel.)
Lorando, N. & Caramaouna, P., 574 (Fev.)
Lorenzo, R. & Boto, D., 213 (Hel.)
Losner, S., with Dubois, 411 (Tryp.)
de Lotto, E., 552 (Y.F.)
Lourie, E. M. & Yorke, W., 404, 405 (Tryp.)
Lowe, J., 39, (51), (340) (Lep.)
—— & Chatterji, S. N., (47) (Lep.)
—— & Greval, S. D. S., (43) (Lep.)
Lucena, D., 60 (Mal.)
Lumsden, W. H. R., with Gordon, 653 (Hel.)
Lund, E. E. & Dennis, E. W., 531 (Misc.)
Lupo, M., with Danielopolu, Craciun & Petresco, 567 *bis* (Fev.)
Lush, D., 848 (Fev.)
Lutz, A., (627) (Lep.)
Lwoff, A., with Ballif, Querido & Ornstein, 435 (Pel.)
——, Querido, A., Digonnet, L. & Garnier, Mlle., 435 (Pel.)
——, with —— & Lataste, 434 (Pel.)
Lwoff, M. & Ceccaldi, J., 13 (Tryp.)

M.

Ma, W. S., with Chung, Khoo & Chang, 119 (Am.)
Maass, E., with Ruge, (43) (Lep.)
McAlpine, J. G., with Smith & Gill, 478 (Hel.)
MacCallan, A. F., 872 (Oph.)
MacCallum, F. O., with Findlay, 93 *bis* (Y.F.)
——, with Murgatroyd & Findlay, 129 (Mal.)
McCammon, W. O., 231 (Misc.)
Macchiavello, A. (254), 254, 255 *bis*, 256, 573 (Fev.), 424, 425 (Pl.)
McClements, S., 590 (Hel.)
MacCreary, D., 380 (Misc.)
McDaniels, H. E., with Rosenfeld, 616 (Rab.)
McDonald, F. G., with Bills & Spies, 445 (Pel.)
Macdonald, G., 379, 526 (Misc.)
Macdonald, I., 118 (Am.)
Macfie, J. W. S., (887) (Misc.)
MacGowan, J. J., with Gotten, 836 (Bl.)
Macheboeuf, M. & Mandoul, R., 648 (Hel.)
Mackay, J. M., 549 (Y.F.)
McKay, W. J. S., 881 (Misc.)
McKendrick, A. G., 616 (Rab.)
McKenzie, A., 219 (Hel.), 530, 809 (Misc.)
McKibbin, J. M., with Waisman, Mickelsen & Elvehjem, (797) (Pel.)
McLendon, S. B., with Young, 362 (Mal.)
Macleod, J. M. H. & Muende, I., 683 (B.R.)
McMahon, J. C., with Wiseman, Symes & Teesdale, 786 (Mal.)
McMullen, W. H., 449 (Oph.)
McNaught, J. B., Beard, R. R. & DeEds, F., 309 (Hel.)
Macvicar, N., 263 (Fev.)
Madras, 273, 274 (Fev.), 277 (Chl.)
de Magalhães, O., 562 (Fev.)
—— & Moreira, J. A., (574) (Fev.)
Magath, T. B. & Meleney, H. E., 729 (Am.)
Maggi, A., with Castex & Orosco, 589 (Hel.)
Maier, E., 461 (Vms.)
Mainzer, F., 433, 434 (Pel.), 485 (Hel.)
—— & Krause, M., 487 (Hel.)
Maister, M. & Miller, A., 260 (Fev.)
Makara, G., with Bodrogi, 114 (Am.)
Malbrant, R., 563 (Fev.), 768 (Leish.)
Maldonado Romero, D., 330 (Lep.)
Malik, K. S., with Lahiri & Das, 718 (Chl.)
——, with Pasricha, Lal & Biswas, 750 (Diet.)
Manalang, C., 48 (Lep.)
Manalang, J., 331, 630 (Lep.)
Manca, S., 503 (Mal.)
Mandekos, A. G., 183 (Mal.)
Mandoul, R., 374 (Dys.)
——, with Macheboeuf, 648 (Hel.)
Manifold, J. A., 174 (Mal.)
Manoilesco, C., with Nitzulesco & Popovici, 65 (Hel.)
Manouélian, Y., 642 (R.B.F.)
Manson-Bahr, P., 174 (Mal.), 535 (B R.)
—— & Ransford, O. N., 99 (Der.)
Manwell, R. D., 60 (Mal.)
—— & Goldstein, F., 60, 508, 794 (Mal.)
—— & Voter, M. A., 509 (Mal.)
Manya, with Bernard, Alcay & Guisoni, (150) (Hel.)
Maplestone, P. A. & Dey, N. C., 97, 98 (Der.)
—— & Mukerji, A. K., 213, 661 (Hel.)
—— & Sundar Rao, S., 307 (Hel.)
Marberg, K., with Reitler & Btesh, 259 (Fev.)
Marchoux, E., 331 (Lep.)
—— & Chorine, V., 49 *bis* (Lep.)
Mareschal, P., with Sparrow, 268, 850 (Fev.)
Mariani, G., 255, 578 (Fev.)
Marill, F., Alcay, L. & Musso, J., 483 (Hel.)
——, with —— & ——, 487 (Hel.)
——, with ——, —— & Castryck, 150, 483 (Hel.)
——, Guily, P. & Kessis, R., 291 (Mal.)
Mario Alonso, L., 586 (Hel.)
Markovich, N. J., 127 (Mal.)
Marliengeas, with Rivoalen, Marty & Saint-Etienne, 584 (Fev.)
Marneffe, H., Gaschen, H. & Nguyen-Ba-Tung, 355 (Mal.)
Marotta, G., 363 (Mal.)
—— & Sandicchi, G., 180 (Mal.)
Marras, F. M., 722 (Chl.)
Martin, D. W., with Smith, 806 (Pel.)
Martin, L. A., with Blanc & Baltazard, 262 (Fev.)
Martin, P. H., with Duncan & Murgatroyd, 96 (Der.)
Martin, R. (347) (Leish.)
——, with Parrot (347) (Leish.)
Martin-Lipmann, Mme., with Nobécourt, 575 (Fev.)
Marty, with Rivoalen, Marliengeas & Saint-Etienne, 584 (Fev.)
Maru, A. M. (444) (Pel.)
Massias, C. & Nguyên-Dinh-Hao, 298 (Hel.)
Masucci, U., 257 (Fev.)
Mata, C., 314 (Bb.)
Mathews, G. B., with Tchang, 579 (Fev.)
Mathis, M., with Lépine & Sautter, 614 (Rab.)
Matsunobu, M., 502 (Mal.)

Mattei, C., Giraud, P., Dumon, G. & Dumon-Lenègre, Mme., 349 (Leish.)
Maxianovitch, M. I., 517 (Vms.)
Mayer, M., 734 (Am.)
Maynard, N. H. (46) (Lep.)
Mayne, B., 286 (Mal.)
Mayrac, F., 853 (Fev.)
Mazumdar, D. C., with Chopra & Roy, 750 (Diet.)
Mazumdar, P. C., with Strickland & Sen Gupta, 177 (Mal.)
Mazumder, A., with Basu, (45) (Lep.)
Mazza, S., 413, 415 (413) (Tryp.)
——, Basso, G. & Basso, R., 715 (Tryp.)
——, ——, —— & Jörg, M. E., 414 (Tryp.)
—— & Cornejo, A., 229 (Misc.)
——, Giordano, J. J. & Dobladez, M. J. L., 714 (Tryp.)
—— & Jörg, M. E., 415 (Tryp.)
—— & Miyara, S., 715 (Tryp.)
——, Tobar, R. G. & Jörg, M. E., (714) (Tryp.)
Mazzotti, L., 142, 712 *bis*, 713 *bis* (Tryp.)
——, with Brumpt, E. & Brumpt, L. C., 141 (Tryp.), 201 (R.F.)
Measham, J. E., 837 (Bl.)
Medical Research Council, 628 (Lep.)
Medina, M., with Celis, 120 (Am.)
Medulla, C., 227 (Misc.)
Megnin, J., with Lartigue, Grenoilleau & Laurens, (366) (Mal.)
Megrail, E., with Doull, 627 (Lep.)
Meiklejohn, A. P. & Kark, R., 434 (Pel.)
de Meillon, B. (658) (Hel.)
—— & Gear, J., 128, 739 (Mal.)
——, with ——, 260, 263 (Fev.)
—— & Holland, E. 480 (Hel.)
—— & Leeson, H. S., (665) (Mal.)
Meléndez, J. F., with Carr & Ros, 664 (Mal.)
Meleney, H. E., with Frye, 115 (Am.)
——, —— & Leathers, W. S., 115 (Am.)
——, with Magath, 729 (Am.)
Mellanby, K., 379 (Misc.)
de Mello, F. & Vaga, A. (372) (Dys.)
Mendelson, R. W., 730 (Am.)
Mendes, E. & Gil de Castro Cerqueira, 336 (Lep.)
Menk, W. & Mohr, W., 377 (Dys.)
Menon, K. P., with Cochrane & Pandit, 331 (Lep.)
Menon, M. K., with Russell & Sweet, 782 (Mal.)
Menon, T. B., 61 (Mal.)
—— & Veliath, G. D., 591 (Hel.)
Meriggi, L., with Tarabini Castellani, 698 (Tryp.)
Mertens, W. K., 279 (Chl.)
—— & Beeuwkes, H., 721 (Chl.)
Mesnard, J. & Toumanoff, C., 564 (Fev.)
Messerlin, A., with Sicault, 862 *bis* (Mal.)
Meyer, K. F., 424 (Pl.)
Michelson, L. & Wilcox, A., 875 (Misc.)
Mickelsen, O., with Waisman, McKibbin & Elvehjem (797) (Pel.)
Milam, D. F., 191 (Rab.)
Miller, A., with Maister, 260 (Fev.)
Miller, M. J., 113 (Am.), 478 (Hel.), 823 (Pl.)
Millet, C. T., 744 (Mal.)
Milliau, M., with Girard, 620 (Rab.)
Minchin, R. L. H., 677 (Misc.)
Mindlin, S. & Urribarri, A., 300 (Hel.)
Mindus, E., 439 (Pel.)
Ministry of Health (571) (Fev.)
Mira, M. G., 658 (Hel.)
Miranda, N., with Talice & Costa (145) (Tryp.)
Mironova, M. N., 153 (Hel.)
Missiroli, A., 671, 672, 795 (Mal.)
Mitchell, R. H., with Shaffer & Shaul, 533 (Misc.)
Miyahara, H., 600 (Mal.)
Miyanaga, S., 489 (Hel.)
Miyara, with Mazza, 715 (Tryp.)
Mochtar, A. & Collier, W. A., 644 (645) (Lept.)
Mohan, B. N., with Russell, 359 *bis*, 741 (Mal.)
Mohr, W., 30 (Sp.)
——, with Menk, 377 (Dys.)
Moiser, B., 633 *bis* (Lep.)
Mol, G., with van Riel, 419 (Pl.)
Molinari, G., (732) (Am.)
Mollaret, P. & Schneider, J., 457 (Mal.)
Momma, K., Yamashita, J. & Kamitani, K., 648 (Hel.)
Monacelli, M., 48 (Lep.)
de Monbreun, W. A., with Anderson, K., 533 (Misc.)
Money, T. D. F., 624 (Lep.)
Monteiro, E. S., 311 (Bb.)
Montel, L. R., Le-Van-Phung, Do-Van-Hoanh & Tran-Van-Hanh, 43 (Lep.)
Montel, M. L. R., 634 (Lep.)
—— & Do-Van-Hoanh, 599 (Mal.)
Montestruc, E., 637 (Lep.)
Monthly Bulletin of the Bureau of Health, Manila, 48 (Lep.)
de Moor, C. E., 279 *bis* (Chl.)
Moore, D. F., 432, 798 (Pel.)
Morales, O. S., 656 (Hel.)
Morales Villazon, N., 641 (R.B.F.)
——, with Savino & Anchezar, 422 (Pl.)
Moreau, P., 829 (Pl.)
Moreira, J. A., with de Magalhães (574) (Fev.)
Morishita, K., 266 (Fev.), 601 (Mal.)
Mornet, P., 768 (Leish.)
Morris, H. H., Hwang, M. S. & Kuo, P. T., 799 (Pel.)
Morris, J. P., with Canton & Rosedale, 887 (B.R.)
Mosna, E., 55, 453, 501 (Mal.)
Motta, O. C., 121 (Am.)
Mourigán, H., with Talice, Osimani & Fernández-Lascano, 145 (Tryp.)
Moussatché, H., with Penna, 558 (Y.F.)
Mozley, A., 483 (Hel.)
Mu, J. W. & Kurotchkin, T. J., 98 (Der.)
Mudrow, L., with Kikuth, 188, 671 *bis* (Mal.)
Muende, I., with Macleod, 683 (B.R.)
Mühlens, P., 571 (Fev.)
Muir, E., 41, 328, 333, 340 (Lep.), 379 (Misc.)
—— & Chatterji, K. R., 328 (Lep.)
——, with Rogers, 603 (B.R.)
Mukerji, A. K., with Maplestone, 213, 661 (Hel.)
Mukerji, B., with Bose & Chopra, 456 (Mal.)
Mukherji, N. N., with Dharmendra, 628 (Lep.)
Murano, G., 770 (Leish.)
—— & Vecchio, F., 348 (Leish.)

Murgatroyd, F., 700 (Tryp.), 848 (Fev.)
——, with Duncan & Martin, 96 (Der.)
——, with Fairley, 834 (Bl.)
——, Findlay, G. M. & MacCallum, F. O., 129 (Mal.)
de Muro, P., 375, 377, 378 (Dys.)
—— & Curatolo, A., 871 (Dys.)
—— & Impallomeni, R., 375 (Dys.)
Murray, D. R. P., 294 (Mal.)
Murray, N. L., 229 (Misc.)
Murrell, T. W., 205 (R.F.)
Musser, J. H., 841 (Fev.)
Musso, J., with Alcay & Marill, 487 (Hel.)
——, with ——, —— & Castryck, 483 (Hel.)
——, with ——, —— & ——, 150 (Hel.)
——, with Marill & Alcay, 483 (Hel.)
Muylle, G., with Rodhain, 61 (Mal.)

N.

Nabarro, D. & Edward, D. G., 362 (Mal.)
Nabokov, V. A., with Serguiev, Zeifert & Kachalova, 506 (Mal.)
Nagle, N., with Sulkin, 193 (Rab.)
Nair, V. G., with Iswariah, 26 (Y. & S.)
Najjar, V. A. & Wood, R. W., 798 (Pel.)
Nakaoka, K. (597) (Hel.)
Napier, L. E., 349 (Leish.)
Narayan, J. S., 342 (Lep.)
Nash, T. A. M., 15, 696 (Tryp.)
Nashed, E., 873 (Oph.)
Nataf, R., with Burnet & Cuénod, 448 (Oph.)
——, with ——, —— & Roussel, 873 (Oph.)
Native Medical Practitioner, Suva, 220 (Misc.)
Nauss, R. W. & Rappaport, I., 728 (Am.)
Neghme Rodriguez, A., 113 *quat.* (Am.), 587 (Hel.)
Negroni, P., 533 (Misc.)
Neill, A., with Greig, 361, 456 (601) (Mal.)
Nguyen-Ba-Tung, with Marneffe & Gaschen, 355 (Mal.)
Nguyên-Dinh-Hao, with Massias, 298 (Hel.)
Nguyen Huu Phiem, with Galliard, 305 (Hel.)
Nguyen-xuan-Nguyen, with Joyeux & Truong-cam-Cong, 216 (Hel.)
Nicholas, W. A., 293 (Mal.)
Nicolesco, P., 571 (Fev.)
Nielsen, H. E., with Lehmann, 313 (Bb.)
Nigeria, 558 (Y.F.), 694 (Tryp.)
Nijs, J., with Gavrilov, 808 (Misc.)
Nikolajev, B. P., 290 (Mal.)
Niño, F. L., 108, 378 (Dys.)
Nishinarita, F., with Kawamura, Kasahara, Toyama & Tsubaki, 269 (Fev.)
Nitzulescu, J., with Ballif, L., Ornstein & Ballif, L. E., 436 (Pel.)
Nitzulesco, V., Manoilesco, C. & Popovici, R., 65 (Hel.)
—— & Nitzulesco, G., 68 (Hel.)
Niven, J. C., 134, 135 (Mal.)
Niven, J. E., 131 (Mal.)
Nobécourt, P. & Martin-Lipmann, Mme., 575 (Fev.)
Nolan, M. O., with Cram, 300 (Hel.)
Noosten, H. H., 881 (Misc.)
Northern Rhodesia, 230, 887 (Misc.)
Noury, M., 621 (Rab.)
Nowicki, E., 139 (Tryp.)
Núñez Andrade, R., 42 (Lep.)
Nyasaland Protectorate, 370 (Bl.), 675 (Misc.)

O.

Oag, R. K., 200 (R.F.)
Oberdoerffer, M. J., (340), 629 (Lep.)
—— & Collier, D. R., 47 (42) (Lep.)
Oberlé, G., with Sicé & Robin, 18 (Tryp.)
O'Brien, A. J. R., 7 (Tryp.), 88, 552 (Y.F.)
Odom, V., with Faust, Sawitz, Tobie, Peres & Lincicome, 62 (Hel.)
——, with Sawitz & Lincicome, 62 (Hel.)
Ogaard, A. T., with Goldsmith & Gowe, 806 (Pel.)
Oh, J., 462 *bis* (Vms.)
Ohama, S., 300 (Hel.)
Ohito, E., with Kubo, 365 (Mal.)
Oldroyd, H., with Edwards & Smart, 387 (B.R.)
Olitzki, L., with Kligler, I. J. & Kligler, H., 710 (Tryp.)
de Oliveira, P., 23 (Y. & S.)
de Oliveira, W., 419 (Pl.)
Olmer, D. & Olmer, J., 845 (Fev.)
—— & Pieri, J., 845 *bis* (Fev.)
Olmes de Carrasco, H., 811 (Misc.)
Omori, N., 204 (R.F.)
O'Neill, J. H., 364 (Mal.)
Ordman, D., 198 (R.F.)
—— & Jones, F. R., 638 (R.F.)
Ornstein, I., with Ballif, Lwoff & Querido, 435 (Pel.)
——, with ——, Nitzulescu & Ballif, L. E., 436 (Pel.)
Orosco, G., with Castex & Maggi, 589 (Hel.)
Ortega, J., 583 (Fev.)
Osaka, K., with Yokogawa, S., Kobayasi, Yumoto, Ro & Yokogawa, M., 651 (Hel.)
Osimani, J. J., with Talice, 141 (Tryp.)
——, with —— & Costa, 145 (Tryp.)
——, with ——, —— & Rial, 889 (B.R.)
——, with ——, Mourigán & Fernández-Lascano, 145 (Tryp.)
Ota, M. & Sato, S., 628 (Lep.)
Otto, G. F., 593 (Hel.)
—— & Abrams, E., 309 (Hel.)
—— & Landsberg, J. W., 593 (Hel.)
Outes, J. D., (824) (Pl.)
Ovalle Sánchez, H., 841 (Fev.)

P.

Pacheco, G. & Péres, J. N., 721 (Chl.)
Packchanian, A., 142 (Tryp.)
Packer, D. M., with Cowdry & Ravold, 50 (Lep.)
Paget, H., Trevan, J. W. & Attwood, A. M. P., 45 (Lep.)
Paige, B., with Wolf & Cowen, 236 (Misc.)
Pallary, P., 485 (Hel.)
Pallister, R. A., 800, 801 (Pel.)
Palomino, J. C., with Sáenz, 39 (Lep.)
Panayotatou, A., 486 (Hel.)

Pandit, C. G., with Cochrane & Menon, 331 (Lep.)
——, Rao, R. S. & Shortt, H. E., 274 (Fev.)
—— & Shortt, H. E., 273 (Fev.)
——, with ——, Anderson & Rao, 853 (Fev.)
Panferova, E. A., with Chub, 130 (Mal.)
Pang, K. H., 261 (Fev.)
——, with Zia & Liu, 579 (Fev.)
Pannekoek-Westenburg, S. J. E. & van Veen, A. G., 315 (Bb.)
Pansini, G., 126, 452 (Mal.)
Paranjothy, J. T., 831 *bis* (Pl.)
Pardal, R., (213) (Hel.)
Parker, R. R., Kohls, G. M., Cox, G. W. & Davis, G. E., 254 (Fev.)
Parrot, L. & Martin, R., (347) (Leish.)
——, with Sergent, Ed., Sergent, Et. & & Catanei, 179 (Mal.)
Pasricha, C. L., with Chopra & Banerjee, 751 (Diet.)
——, with —— & Lal, 588 (Hel.)
——, Lahiri, M. N., Das, P. C. & Paul, B. M., 276 (Chl.)
——, Lal, S. & Banerjee, K., 749 (Diet.)
——, ——, Malik, K. S. & Biswas, P. K., 750 (Diet.)
Passmore, R., with Aykroyd & Krishnan, 431 (Pel.)
Patel, T. B., 840 (Fev.)
Patiño Camargo, L., 271, 582 (Fev.)
Paul, B. M., with Pasricha, Lahiri & Das, 276 (Chl.)
Paulino, P., with Concepcion, Camara & Gargaritano, 755 (Bb.)
Paunescu-Podeanu, A. & Carangiu, O., 504 (Mal.)
Pearce, L., 20 (Tryp.)
Pearce, R., 328 (Lep.)
Pearson, Y., with Dubois, 199 (R.F.), 206 (R.B.F.)
Pedersen, H., with Jordan & Shu, 192 (Rab.)
Peel, E., with van Hoof &Henrard, 703 *quart.* (Tryp.)
Peltier, M., Durieux, C., Jonchère, H. & Arquié, E., 92, 557 (Y.F.)
Penna, H. A., 558 (Y.F.)
—— & Moussatché, H., 558 (Y.F.)
Perdomo Hurtado, B., 30 (Sp.)
Pereira, J., 818 (Misc.)
Peres, C., with Faust, Sawitz, Tobie, Odom & Lincicome, 62 (Hel.)
Péres, J. N., with Pacheco, 721 (Chl.)
Pérez, M., with Earle, Del Rio & Arzola, 178 (Mal.)
Perris, E. P., 837 (Bl.)
Perry, H. M., 174 (Mal.)
Peruchena, J. G., with Fuertes, 334 (Lep.)
Pessôa, S. B. & de Barros, N. V., 143 (Tryp.)
——, with Pestana, 776 (Leish.)
——, with Villela & Pestana, 353 (Leish.)
Pestana, B. R. & Pessôa, S. B., 776 (Leish.)
——, with Villela & Pessoa, 353 (Leish.)
Petit, J., 447 (Oph.)
Petresco, M., with Danielopolu, Lupo & Craciun, 567 *bis* (Fev.)
Pfeiffer, D. H., 574 (Fev.)
Philip, C. B., (234) (Misc.)
—— & Davis, G. E., 640 (R.F.)
Philps, A. S., 873 (Oph.)
Piaggio-Blanco, R. A. & Garcia-Capurro, F., 589 (Hel.)
Piccaluga, L., 358 (Mal.)
Pieri, J., with Olmer, 845 *bis* (Fev.)
Pifano, F., with Lent, 142 (Tryp.)
Pilgerstorfer, E., with Grüneis, 869 (Dys.)
Pinard, M. & Brumpt, L. C., 18 (Tryp.)
Pinkerton, H. & Bessey, O. A., 268 (Fev.)
Pinotti, M., Genofre, W., Romeu, L. B. & Viana, M., 824 (Pl.)
Pinto, C., 498 (Mal.)
Piorkowski, F. O., 457 (Mal.)
Pirumov, K. N. & Ananjan, S. A., 275 (Fev.)
Pistone, F., (559) (Y.F.)
Pizzillo, G., 176, 364, 602 (Mal.)
Plantilla, F. C., with Rodriguez & Wade, 630 (Lep.)
Platts, N. G., with Hull & Dove, 235 (Misc.)
Plotnikov, N. N., 152 (Hel.)
Plotz, H., with Zinsser & Enders, 580 (Fev.)
Pluchon, J. P., 340 (Lep.)
Plunkett, O. R. L. L., 432 (Pel.)
Pock-Steen, P. H., 31 (Sp.)
Pocoule, A., with Gaud, 565 (Fev.)
Poleff, L., 447 (Oph.)
Poleshko, G. V., with Konj, 506 (Mal.)
Polidori, V., 221 (Misc.)
Pollet, L., with Duvoir, Brumpt & Chénebault. 596 (Hel.)
Pollock, R. C., 659 (Hel.)
Ponnambalam, C., 464 (Vms.)
Pons, J. A., 841 (Fev.)
Popotas, C., 772 (Leish.)
Popovici, R., with Nitzulesco & Manoilesco, 65 (Hel.)
Porto, J. & de Soldati, L., 754 (Bb.)
Possolo, H., (45) (Lep.)
Poudevigne, H., with Sicé & Berthon, 22 (Y. & S.)
Powers, G. E. & Headlee, T. J., 94 (Y.F.)
Prat, D. & Lopez-Gutierrez, J. C., 590 (Hel.)
Prawirohardjo, S., 304 (Hel.)
——, with Sandground, 153 (Hel.)
Prendel, A. R. & Somov, A. A., 127 (Mal.)
Proca, G. & Bobes, S., 618 (Rab.)
Prommas, C., 883 (Misc.)
Prost, P., with Farinaud, 289, 494 (Mal.)
Provost, A., with Roubaud, 139, 399 (Tryp.)
Prudhomme, R. O., (Lep.)
Public Health Reports, 517 (Vms.)
Public Works, 505 (Mal.)
Puduval, T. K., with Wats, 827 (Pl.)
——, with —— & Wagle, 420 (Pl.)
Puntoni, V., 620 (Rab.)
Pupo, J. A., 633 (Lep.)

Q.

Querido, A., with Ballif, Lwoff & Ornstein, 435 (Pel.)
——, with Lwoff, Digonnet & Garnier, Mlle., 435 (Pel.)
——, —— & Lataste, C., 434 (Pel.)
Quinby, G. E., with Bang & Simpson, 742 (Mal.)

R.

Rachmilewitz, M., with Adler, 776 (Leish.)
Radaody-Ralarosy, P., with Girard, 830 (Pl.)
Radlo, P. & Rostowski, L., 448 (Oph.)
Radna, R., 38 *bis*, 45, 47, 336, 337, 342 (Lep.)
——, with Dubois, (43) (Lep.)
Rafeal Risquez, J., (149) (Hel.)
Raffaele, G., 285 (Mal.)
Ragiot, C., with Alain, 123 (Am.)
Raj, M. P., with Cochrane & Roy, 342 (Lep.)
Rajam, R. V., 25 (Y. & S.)
—— & Rangiah, P. N., 731 (Am.)
Ram, T., 883 (Misc.)
Raman, T. K., 803 (Pel.)
Rames, C., 351 (Leish.)
Ramírez Enríquez, F., 516 (Vms.)
Rangiah, P. N., with Rajam, 731 (Am.)
Ransford, O. N., with Manson-Bahr, 99 (Der.)
Rao, M. S., 825, 826 (Pl.)
Rao, R. S., with Pandit & Shortt, 274 (Fev.)
——, with Shortt, Pandit & Anderson, 853 (Fev.)
Rao, S. R., 418 (Pl.)
Rao, S. S., with Basu, 301 (Hel.)
——, with Chopra, 656 (Hel.)
Rappaport, I., with Nauss, 728 (Am.)
Rashina, M. G. & Khovanskaya, A. I., 133 (Mal.)
Raskine, A. J., 746 (Mal.)
Ravold, A., with Cowdry & Packer, 50 (Lep.)
Raymond, (843) (Fev.)
Raymond, W. D., 72 *bis*, 679 (Misc.)
Raynal, J., 569 (Fev.)
—— & Fournier, J., 256 (Fev.)
——, —— & Velliot, E., 256 (Fev.)
—— & Lieou, Y. C., 614, 821 (Rab.)
——, —— & Feissolle, L., 720 (Chl.)
Raynaud, M., with Le Renard, 258 (Fev.)
Rebagliati, R., (584) (Fev.)
Reddy, D. G. & Subramaniam, R., 349 (Leish.)
Redmond, W. B., 58 (Mal.)
Reenstierna, J., 629 (Lep.)
Reeves, J. L., with Behrens, Schweiger & Barker, 195 (Rab.)
Reichenow, E., 14 (Tryp.)
Reid, J. D., Scherer, J. H. & Irving, H., 817 (Misc.)
Reis, K., 872 (Oph.)
Reitler, R., Btesh, S. & Marberg, K., 259 (Fev.)
Remlinger, P. & Bailly, J., 615 *bis*, 616 (Rab.)
Renault, L., with Versiani, 214 (Hel.)
Repond, A., 675 (Misc.)
Resseler, R., with Dubois, 340 (Lep.)
Revue Internationale du Trachome, 447 (Oph.)
Reyer, W., 235, 817 (Misc.)
Reynes, V., with Delbove, 573 (Fev.)
—— & Richard, J., 578 (Fev.)
Reynolds, F. H. K., with Stone, 360 (Mal.), 532 (Misc.)
Rhodes, A. J., 448 (Oph.)
Rial, B., with Talice, Costa & Osimani, 889 (B.R.)
Richard, J., with Reynes, 578 (Fev.)
Richards, P. W., Tansley, A. G. & Watt, A. S., 699 (Tryp.)
Richet, C. & Antoine, G., 412 (Tryp.)
Richet, P., 806 (Hel.)
Ricklin, J., 281 (Misc.)
van Riel, J. & Moi, G., 419 (Pl.)
Riley, W. A., 587 (Hel.)
Riou, M., 813 (Misc.)
——, with Toullec, 31 (Sp.)
——, with Vogel, 8 (Tryp.), 67 (Hel.), 91 (Y.F.), 125 (Mal.), 196 (Rab.)
Rishi, D. P., 339 (Lep.)
Risquez, J. R., 104 (Der.)
Ristorcelli, A., 347 (Leish.)
Rita, G., 191 (Rab.)
de Ritis, F., 794 (Mal.)
Ritsert, K., 435 (Pel.)
Rivoalen, Bruneau & Kernevez, 578 (Fev.)
——, Marliengeas, Marty & Saint-Etienne, 584 (Fev.)
Rivoalen, A., (306) (Hel.)
Rivoalen, P., with Beaudiment, 340 (Lep.)
Rizzo, J., 732 (Am.)
Rizzotti, G., 644 (Lept.)
Ro, M., 490 (Hel.)
——, with Yokogawa, S., Kobayasi, Yumoto, Osaka & Yokogawa, M., 651 (Hel.)
Roberts, F. H. S., 597 (Hel.), 844 (Fev.)
Roberts, J. I., 260 (Fev.), 875 (Misc.)
——, with Anderson, 884 (Misc.)
Robertson, D. S., (441) (Pel.)
Robertson, R. C., 792 (Mal.), 810 (Misc.)
Robin, C. & Brochen, L., 402 (Tryp.)
——, with Sicé & Oberlé, 18 (Tryp.)
Robinson, W. D., with Field, 440 (Pel.)
Rocchi, F., 314 (Bb.), 455 (Mal.)
Rockefeller Foundation, 75 (B.R.)
Rodallec, B., with Sicé, 551 (Y.F.)
Rodhain, J., 367, 669, 670 (Mal.)
—— & van Hoof, T., 795 (Mal.)
—— & Lassman, P., 459, 509, 794 (Mal.)
—— & Muylle, G., 61 (Mal.)
Rodriguez, J. N., 627 (Lep.)
—— & Wade, H. W., 338 (Lep.)
——, with ——, 338 (Lep.)
——, —— & Plantilla, F. C., 630 (Lep.)
——, with —— & Tolentino, 631 (Lep.)
Rodriguez-Molina, R., 529 (Misc.)
Roe, M. A., Lillie, R. D. & Wilcox, A., 87[illegible] (Misc.)
Roetti, C., (234) (Misc.)
Rogers, L., 31 (Sp.), 350 (Leish.)
—— & Muir, E., 603 (B.R.)
Rogers, W. P., (592) (Hel.)
Rohrschneider, W., 447 (Oph.)
Roman, E., 298 (Hel.)
Romaña, C., 143, 416 (Tryp.)
——, with Colas-Belcour (347) (Leish.)
Romaña, J., with Roubaud, 413 (Tryp.)
Romeu, L. B., with Pinotti, Genofre & Viana, 824 (Pl.)
Ros, A., with Carr & Meléndez, 664 (Mal.)
Rosedale, J. L., 752 (Bb.)
——, with Canton & Morris, 887 (B.R.)
Rosenblum, L. A., with Jolliffe & Fein, 43[illegible] (Pel.)
Rosenfeld, I. & McDaniels, H. E., 61[illegible] (Rab.)
Rosenfeld, S. & Glass, S., 511 (Vms.)
Rosenthal, E., 185 (Mal.)
Rosier, H. J., 425 (Pl.)

Ross, H. 89, 632 (Leg.)
—— with Blatch. 42 (Leg.)
Rostkowski, L. with Fedor. 448 (Ophth.)
Rotberg, A. 46 (Leg.)
Roux, E. 669 (Hel.)
Rovin, 466 (Fev.)
Rowland, H. & Desnoeuxaux, J. (317) (Hel.)
—— & Provost, A. 184, 396 (Typ.)
—— & Romada, J. 419 (Typ.)
Royesse, H., with Bonner, Gwenod & Nenad 673 (Ophth.)
Row, 48 (Leg.)
Roy, A. C., with Chopra & Mazumder 751 (Dys.)
Roy, D. N. 500 (Mal.)
—— with Chopra, Hayyar & Sen. 746 (Mal.)
—— with Strickland & Sen Gupta. 586 (Mal.)
Roy, E. with Cochrane & Raj, 842 (Leg.)
Roy, S. C. 291 (Mal.)
Rubegni, R. 582 (Hel.)
Ruddock, J. C. & Hope, R. B. 584 (Misc.)
Ruge & Maass, E. 45 (Leg.)
Ruge, H. 259 (Fev.)
di Ruggero, with Jolly 298 (Fev.)
Rust, E. with Kamm, 857 (Mal.)
Ruhe, A. M., with Kolmer & Kast. 708 (Typ.)
Russell, A. J. H. 91 (Y.F.)
Russell, P. F. & Jacob, V. F., 53 (Mal.) 506 sq. (Mal.)
—— & Knipe, F. W. 504 (Mal.)
—— & Molner, R. N., 359 bis, 741 (Mal.)
—— Sweet, W. C. & Manson, M. E. 782 (Mal.)
Ross, E. 422 (Pl.)
Rutherford, R. N. 464 (Vims.)
Ryan, G. A. 887, 686 (Leg.)

S.

Sabrai, B. & Pelkmans, J. C. 39 (Leg.)
Sacaz Vera, C. 425, 824 (Pl.)
Saghaan, T. 690 (Mal.)
Saldanyi Hearts Project, 625 bis (Leg.)
Saint-Etienne, with Bouvalem, Marchengeas & Mikry 584 (Fev.)
Salamaka, T. 571 (Dys.)
—— & Hirata, T. 571 (Dys.)
Salehan, 448 (Pl.)
Salerno, G. 196 (Rab.) 258, 288 (Fev.) 408 (Typ.) 559 (Y.F.)
Salm, H. 442 (Pel.)
Salvesugh, F. J., with Costa, 516 (Vims.)
Salvesen, O. 498 (Pl.)
Samara, M. with Le Gac & Servant 273 (Fev.)
Sanders, J. F. & Dawson, W. T. 185 (Mal.)
Sandgrovend, J. H. 158, 819 (Hel.)
—— with Bonne, 487, 491 (Hel.)
—— & Prawirohardjo, S. 158 (Hel.)
Sandholm, G., with Marrera, 180 (Mal.)
Sanjuan, A. 640 (Leg.)
San Pedro, F. 162 (Mal.)
Sapery, J. J. 729 (Am.)
—— & Johnson, C. M., 111 (Am.)
Sardjito, 882 (Leg.)
—— with Assten Tabar Berkens & Dyotikana. 788 (Lept.)
—— with Hardjosoy 885 (Misc.)
Sarkar, S. L. 746 (Dys.)
—— & Bhattacharyya, B. M. 47 (Leg.)
Sasportas, L. 697 (Mal.)
Sauf, M. H. with Kirk, 771 (Leish.)
Saxe, S. with Ova, 828 (Leg.)
Saxon, E. 356 (Mal.)
Saunders, G. 397 (Typ.)
Sauvage, J. 196 (R.F.)
—— with Joyeux, 644 (Leish.)
——, with —— & Sadé, 298 (Mal.)
——, with Sicé & Eldes, 667 (Mal.)
Sautter, V., Mlle., with Lemée & Marnas 614 (Rab.)
Savagnone, L. 191 (Rab.)
Savino, E. & Ancheszar, B. 426 (Pl.)
——, Morales Villazon, N. & Ancheszar, B. 426 (Pl.)
Savour, S. E., with Lewiswarn, 676, 647 (Fev.)
Sawon, W. 306 (Hel.)
—— with Faust, Tobie, Irons, Perez & Lincoln 40 (Hel.)
——, Odom, V. L. & Lincoln, D. B., 41 (Hel.)
Sawyer, W. A. (558) (Y.F.)
Sazana, G. S. 850 (Leish.)
Scalibi, V., Jr. 108 (Am.), 196 (R.F.), 205 (R.B.F.)
Schaafsma, A., with Gesser, 913 (Vims.)
Schaaff, J. W. (198) (Mal.)
Scherer, J. H., with Reed & Irving, 687 (Misc.)
Schiavi, C. 150 (Hel.)
Schilling, C. 866 bis, 867, 790 (Mal.), 709 (Typ.)
Schlaiber, E. M. 924 (Misc.)
Schlenk, F. with v. Eiser 446 (Pe.)
Schmidt, H. L. Jr. with Schmamacker Jessen & Weaver 487 (Pe.)
Schneider, J. with Devoint & Bellot 186 (Mal.)
——, with —— & Kernet-Grosmier 456 (Mal.)
——, with Mollaret 457 (Mal.)
Schneideber, A. W. 721 (Am.)
Schnerer, M., with Lewison & Devignat 826 (Pl.)
Schreczenmayr, A. 673, 877 (Misc.), 753 (Hel.)
Schujman, S. 39, 337 (Leg.)
Schulemann, W. 282 (Misc.)
Schwarz, J. & Strauch, M. 649 (Hel.)
Schwenger, L. B., with Behrens, Barker & Reeves, 195 (Rab.)
Scott, C. J. 21 (Y. & S.)
Scott, H. E. 867 (Misc.)
Scott, J. A. 484, 647 (Fev.)
Scott, G. 180 (Mal.)
Seal, S. C. 228 (Cil.)
Sebrell, W. H. & Butler, R. E. 436 (Pel.)
Sen, M. 301 (Pe.)
Selvyn-Clarke, 618 (Misc.)
Sen, B., with Chopra, Das Gupta & Ahmed 577 (Dys.)
——, with ——, —— & Hayyar 186 (Mal.)
Sen, B. with Chopra, Roy & Hayyar 746 (Mal.)
——, with —— & Hayyar, 503, 746 (Mal.)
Sen, P., 365 (Mal.)
Sen, S., with Chopra & Basu, 196 (R.B.F.)

Senekji, H. A., 351 *bis*, 779 *bis* (Leish.), 726 (Am.).
—— & Beattie, C. P., 587 (Hel.)
——, Boswell, C. & Beattie, C. P., 479 (Hel.)
Senevet, G., (553) (Y.F.)
—— & Abonnenc, E., (95) Y.F.)
Sen Gupta, S. C., with Strickland & Mazumdar, 177 (Mal.)
——, with —— & Roy, 859 (Mal.)
Sergent, E., (176) (Mal.), 519 (Vms.)
Sergent, Ed., Sergent, Et., Parrot, L. & Catanei, A., 179 (Mal.)
Sergent, Et., 187 (Mal.)
——, with Sergent, Ed., Parrot & Catanei, 179 (Mal.)
Serguiev, P. G., Nabokov, V. A., Zeifert, J. A. & Kachalova, E. K., 506 (Mal.)
Serra, G., with Lenti, 23 (Y. & S.)
Servant, J., with Le Gac, 18, 273 (Tryp.)
——, with —— & Samara, 275 (Fev.)
Seyberlich & Lê-Thi-Van, 814 (Misc.)
Shaby, J. A., with Hoff, 147, 592 (Hel.)
Shaffer, F. J., Shaul, J. F. & Mitchell, R. H., 533 (Misc.)
Shah, S. R. A., 232 (Misc.)
Sharma, L. R., 840 (Fev.)
Shastry, T. S., 212 (Hel.)
Shaul, J. F., with Shaffer & Mitchell, 533 (Misc.)
Shchurenkova, A. I. & Dolmatova, A. V., 381 (Misc.)
Shelley, H. M., 675 (Misc.)
Shields, J., 192 (Rab.)
Shields, S. E., with Hull, 380 (Misc.)
Shiga, H., 871 (Oph.)
Shih, H. E., Wu, Y. K. & Lieu, V. T., 122 (Am.)
Shrimpton, E. A. G., with Andrews, 237 (Misc.)
Shortt, H. E., 273, 274 (Fev.), 277 (Chl.)
——, Pandit, C. G., Anderson, W. M. E. & Rao, R. S., 853 (Fev.)
——, with ——, 273 (Fev.)
——, with —— & Rao, 274 (Fev.)
Shu, S., with Jordan & Pedersen, 192 (Rab.)
Shulov, A., 520 (Vms.)
Shute, D. S., (593) (Hel.)
Shute, P. G., 289, 666 (Mal.)
——, with Hutton, 456 (Mal.)
——, with Sinton, 204 (R.F.)
——, with —— & Hutton, 54, 55, 183 (Mal.)
Sicault, G. & Messerlin, A., 862 *bis* (Mal.)
Sicé, A., 705 (Tryp.), 878 (Misc.)
——, with Joyeux & Sautet, 286 (Mal.)
——, Poudevigne, H. & Berthon, H., 22 (Y. & S.)
——, Robin, C. & Oberlé, G., 18 (Tryp.)
—— & Rodallec, B., 551 (Y.F.)
——, Sautet, J. & Ethes, Y., 857 (Mal.)
—— & Torresi, F., 9 (Tryp.)
Siegenbeek van Heukelom, A., 745 (Mal.)
Sikorav, H., with Cain, 377 (Dys.)
Silva, R., with Castaneda, 259 (Fev.)
Silva-Correa, M. R., 227 (Misc.)
Simmons, J. S., with Watson & Faust, (53) (Mal.)
Simons, J., with Joranson, 122 (Am.)
Simpson, T., 21 (Y. & S.)
Simpson, T. W., with Bang & Quinby, 742 (Mal.)
Singh, G., with Ahuja, 277 (Chl.)
Singh, J. & Singh, H., 190 (Mal.)
Sinton, J. A., 56, 57 *bis*, 497, 667, 862 (Mal.)
——, Hutton, E. L. & Shute, P. G., 54, 55, 183 (Mal.)
—— & Shute, P. G., 204 (R.F.)
Sivalingham, V., (292) (Mal.)
Skrjabin, K. I., 662 (Hel.)
Slamet Sudibyo, R. M., 25 (Y. & S.)
Smart, J., 380 (Misc.), (658) (Hel.), 885 (Misc.)
——, with Edwards & Oldroyd, 387 (B.R.)
——, with White & Aubertin, 468 (B.R.)
Smetana, H., 68 (Hel.)
Smillie, W. S., 219 (Hel.)
Smith, C. E., 882 (Misc.)
Smith, D. J. W., 851 (Fev.)
——, with Burnet, Freeman & Derrick, 253 (Fev.)
——, with Derrick, 851 (Fev.)
Smith, E. C., 77 (B.R.)
Smith, H. H., 551 (Y.F.)
Smith, J. E., with Julianelle, 872 (Oph.)
Smith, R. O. A., Halder, K. C. & Ahmed, I., 769 (Leish.)
Smith, S., (186) (Mal.)
Smith, S. G. & Martin, D. W., 806 (Pel.)
Smith, W. H. Y., Gill, D. G. & McAlpine, J. G. 478 (Hel.)
Sneath, P. A. T., 90 (Y.F.)
Sobky, M. F., with Halawani, 864 (Mal.)
Sociedad Argentina Patología Regional, 238 (B.R.)
Soerono, 426 (Pl.)
Sohier, R. & Jaulmes, C., 114 (Am.)
Sokhey, S. S., 28 (Sp.), (48) (Lep.), 292 *bis* (Mal.), 420, 825 (Pl.)
——, Chitre, G. D. & Gokhale, S. K., 427 (Pl.)
—— & Dikshit, B. B., 828 (Pl.)
Solarino, G., 117 (Am.), 281 (Chl.)
Soldati, L., with Porto, 754 (Bb.)
Solomon, H. C., with Kopp, 705 (Tryp.)
Somogyi, J. C., 190 (Mal.)
Somov, A. A., with Prendel, 127 (Mal.)
Soparkar, M. B., 277 (Chl.)
Sorel, (49) (Lep.)
Sorel, F. P. J., 88 (Y.F.)
Sorsby, A., 447 (Oph.)
de Sousa, M., 458 (Mal.)
Southwell, T., with Blacklock, 820 (B.R.)
de Souza, A. R., 41 (Lep.)
Spadaro, O., 869 (Dys.)
Spalding, J. E., with Failmezger, 308 (Hel.)
Sparrow, H., 267 (Fev.)
——, with Durand, 572, 849 (Fev.)
——, with —— & Giroud, 849 (Fev.)
—— & Mareschal, P., 268, 850 (Fev.)
Speziale, V. & Berger, R., (213) (Hel.), 345 (Leish.)
Spies, T. D., 447 (Pel.)
——, with Bills & McDonald, 445 (Pel.)
——, with Frostig, 443 (Pel.)
——, with Lewy & Aring, 805 (Pel.)
——, with ——, Himwich & Frostig, (444) (Pel.)
——, with Vilter, 444 (Pel.)
——, —— & Ashe, W. F., 429 (Pel.)

Spining, W. D., 872 (Oph.)
Spreng, A., 30 (Sp.)
Sproule, J. C., 148 (Hel.)
Stannus, H. S., 430, (431) (Pel.)
Starkoff, O., 65 (Hel.)
Stefanopoulo, G. J., with Kolochine-Erber, 643 (Lept.)
Stein, A. A., 38, 334, 631 (Lep.)
Steinfeld, F. U., 698 (Tryp.)
Stephenson, A. G., 386 (Misc.)
Sternberg, G., 371 (Dys.)
Stiles, C. W., (219) (Hel.)
Still, R. M. L., 816 (Misc.)
Stone, W. S. & Reynolds, F. H. K., 360, (Mal.), 532 (Misc.)
—— & Thompson, A. T., 711 (Tryp.)
Strangways-Dixon, D., 867 (Mal.)
Straub, M., with Schwarz, 649 (Hel.)
Streef, G. M., with Zainal & Streef-Spaan, 592 (Hel.)
Streef-Spaan, A. M., with Zainal & Streef, 592 (Hel.)
Strickland, C., 126, 599 (Mal.)
—— & Baird, S. Y., 129 (Mal.)
——, Roy, D. N. & Sen Gupta, S. C., 859 (Mal.)
——, Sen Gupta, S. C. & Mazumdar, P. C., 177 (Mal.)
Strong, R. P., (564) (Fev.)
Stuart-Bunning, G. H., 379 (Misc.)
Stubbs, T. H., with Young & Coatney, 783 (Mal.)
Suárez, R. M., 28 (Sp.)
Suárez, P. A., 423 (Pl.)
Subramaniam, R., with Reddy, 349 (Leish.)
Sulkin, S. E. & Nagle, N., 193 (Rab.)
—— & Willett, J. C., 194 (Rab.)
Sun, C. J., 775 *bis* (Leish.)
—— & Chang, S., 775 (Leish.)
Sundar Rao, S., with Maplestone, 307 (Hel.)
Surraco, C., 386 (Misc.)
Svensson, R., 756 (B.R.)
Swales, W. E., 69 (Hel.)
Swaminathan, M., with Aykroyd, 797 (Pel.)
Swartzwelder, J. C., 62, (210), 660 (Hel.), 116 (Am.)
Sweet, W. C., with Russell & Menon, 782 (Mal.
Syddiq, M. M., 365 (Mal.)
Sydenstricker, V. P., with Cleckley & Geeslin, 442 (Pel.)
——, Geeslin, L. E., Templeton, C. M. & Weaver, J. W., 439 (Pel.)
——, Schmidt, H. L., Jr., Geeslin, L. E. & Weaver, J. W., 437 (Pel.)
Symes, C. B., with Wiseman, McMahon & Teesdale, 786 (Mal.)

T.

Taborisky, J., 871 (Oph.)
Taddia, L. & Viero, G., 793 (Mal.)
Takita, J., 281 (Chl.)
Taliaferro, W. H., (887) Misc.)
Talice, R. V., 144 (Tryp.)
——, Costa, R. S. & Osimani, J. J., 145 (Tryp.)
——, ——, Rial, B. & Osimani, J., J. 889 (B.R.)
—— & Fuente-Méndez, R. (145) (Tryp.)
Talice, Miranda, N. & Costa, R. S., (145) (Tryp.)
——, Mourigán, H., Osimani, J. J. & Fernández-Lascano, F., 145 (Tryp.)
—— & Osimani, J. J., 141 (Tryp.)
Tang, C. C., 308 (Hel.)
Tanguy, Y., 38 (Lep.)
Tansley, A. G., with Richards & Watt, 699 (Tryp.)
Tappeiner, S., (39) (Lep.)
Tarabini Castellani, G. & Meriggi, L., 698 (Tryp.)
Tasque, P., with Floch & Goerger, 645 (Lept.)
Tattersfield, F., (235) (Misc.)
Tatum, A. L., with Longley & Clausen, 12 (Tryp.)
Taylor, H. W. Y., 114 (Am.)
Tchang, J. & Mathews, G. B., 579 (Fev.)
Tchernomoretz, I., with Adler, 775 (Leish.)
Teesdale, C., with Wiseman, Symes, & McMahon, 786 (Mal.)
Tempel, D., with Fiebiger (211) (Hel.)
Templeton, C. M., with Sydenstricker, Geeslin & Weaver, 439 (Pel.)
Tenhaeff, C. & Ferwerda, S., 591 (Hel.)
Thambiah, S. (44) (Lep.)
Thayer, K. H. (641) (R.F.)
van Thiel, P. H., (454) (Mal.)
Thiodet, M., (218) (Hel.)
Thomas, A. D., 621 (Rab.)
Thomas, R. (293) (Mal.)
Thompson, A. T., with Stone, 711 (Tryp.)
Thonnard-Neumann, E. (601) (Mal.)
Thygeson, P., 872 (Oph.)
Timpano, P., 592 (Hel.)
Timrot, S. D., with Vinogradskaya, 180 (Mal.)
Tjiook Kiem Bok, 78 (B.R.)
Tobar, R. G., with Mazza & Jörg (714) (Tryp.)
Tobie, J., with Faust, Sawitz, Odom, Peres & Lincicome, 62 (Hel.)
Todd, K. W., 25 (Y. & S.), 223 (Misc.)
Tokuyama, S., 209 (Lept.)
Tolentino, J. G., with Wade & Rodriguez, 631 (Lep.)
Tomb, J. W., 276 (Chl.)
Topping, N. H., 264, 564, 575 (Fev.)
—— & Dyer, R. E., 843 (Fev.)
——, with Lillie & Dyer, 572 (Fev.)
Torresi, F., with Sicé, 9 (Tryp.)
Tötterman, G., 215 (Hel.)
Toullec, F. & Riou, M., 31 (Sp.)
Toumanoff, C., 288 (Mal.)
——, with André 287 (Mal.)
—— & Canet, J., 860 (Mal.)
——, with Genevray & Try, 293, 664 (Mal.)
—— & Hoang-Tich-Try, 187 (Mal.)
——, with Mesnard, 564 (Fev.)
——, Try, H. T. & Chang, T. L., 651 (Hel.)
Townsend, R. S., with Lall, 384 (Misc.)
Toyama, T., with Kawamura, Kasahara, Nishinarita & Tsubaki, 269 (Fev.)
Trager, W., 233 (Misc.)
Transactions of the IIIrd Trans-Caucasian Congress for the Campaign against Malaria and Other Tropical Diseases, Baku, 1936 (738) (Mal.)
Tran-Van-Hanh, with Montel, Le-Van-Phung & Do-Van-Hoanh, 43 (Lep.)
Travassos, J. & Dias, E., 574 (Fev.)

Treillard, M., 599 (Mal.)
Trethewie, E. R., (463) (Vms.)
Trevan, J. W., with Paget & Attwood, 45 (Lep.)
Tribedi, B. P. & De, M. N., 749 (Diet.)
——, with —— 232 (Misc.)
Trifilò, N., 777 (Leish.)
Trolli, G., 521 (Misc.)
Trowell, H. C., 431 (Pel.), 883 (Misc.)
Truong-cam-Cong, with Joyeux & Nguyen-xuan-Nguyen, 216 (Hel.)
Try, H. T., 501 (Mal.)
——, with Genevray & Toumanoff, 293, 664 (Mal.)
——, with Toumanoff & Chang, 651 (Hel.)
Tsubaki, S., with Kawamura, Kasahara, Toyama & Nishinarita, 269 (Fev.)
Tsuchiya, H., 117 (Am.)
Tubangui, M. A. & Africa, C. M. (154) (Hel.)
Turner, O., 877 (Misc.)
Turvey, S. E. C., with Cleveland, 186 (Mal.)
Tutumi, S., with Kobayasi, Yokoi & Kawabe, 856 (Mal.)
Tyzzer, E. E. & Weinman, D., 272 (Fev.)

U.

Ueda, M., with Kawamura, 269 (Fev.)
Uganda Protectorate, 397 (Tryp.)
Umboh, T. E. W., with Kraneveld, 412 (Tryp.)
Ungo-Mugdan, A., 459, 672 (Mal.)
Urabe, K., 802 (Pel.)
Urribarri, A., with Mindlin, 300 (Hel.)

V.

Vaga, A., with de Mello (372) (Dys.)
Valaoras, V. G., with Livadas & Canellakis, 291 (Mal.)
Van den Berghe, L., 92, 94 (Y.F.), 482 (Hel.)
——, with van Bogaert, 802 (Pel.)
—— & Kovacs, L., 129 (Mal.)
Van den Branden, 138, 410, 705 (Tryp.)
—— & Geens, J., 237 (Misc.)
Van der Walle, N., 383 (Misc.)
Vanni, V., 352 ,779 (Leish.)
Vanselow, J.-D., (791) (Mal.)
Vargas, A., 743 (Mal.)
—— & Freire, F., 784 (Mal.)
Vargas, L., 360 (743), (744) (789) (Mal.)
Varlakov, M. N. (299) (Hel.)
Vartan, C. K. & Discombe, G., 865 (Mal.)
Vassallo, S. M., 534 (Misc.)
Vazquez-Colet, A. & Africa, C. M. (492) (Hel.)
Vecchio, F., 350 (Leish.)
——, with Murano, 348 (Leish.)
van Veen, A. G., with Pannekoek-Westenburg, 315 (Bb.)
Véghelyi, P., 375, 376 *bis*, 870 (Dys.)
Veliath, G. D., with Menon, 591 (Hel.)
Velliot, E., with Raynal & Fournier, 256 (Fev.)
Venhuis. W. G., 455, 667 (Mal.)
Venkatraman, K. V., 277 (Chl.)
Versiana, W. & Renault, L., 214 (Hel.)
Vespoli, M., 341 (Lep.)
Viana, M., with Pinotti, Genofre & Romeu, 824 (Pl.)
Vieira, J. P., (635) (Lep.)
Viero, G., with Taddia, 793 (Mal.)
Villagrán, R., (295) (Mal.)
Villela, F., 352 (Leish.)
——, Pestana, B. R. & Pessoa, S. B., 353 (Leish.)
Vilter, R. W. & Spies, T. D., 444 (Pel.)
——, with —— & Ashe, 429 (Pel.)
Vinogradskaya, O. N. & Timrot, S. D., 180 (Mal.)
Virnich, A., 734 (Am.)
Vitale, S., with Dubois, 306 (Hel.)
Vladesco, R., with Bertrand, 463 *ter* (Vms.)
de Vogel, W., 723 (Chl.)
—— & Riou, M., 8 (Tryp.), 67 (Hel.), 91 (Y.F.), 125 (Mal.), 196 (Rab.)
Voter, M. A., with Manwell, 509 (Mal.)
Vryonis, G., 360 (Mal.)

W.

Wade, H. W., 336 (Lep.)
—— & Rodriguez, J. N., 338 (Lep.)
——, with ——, 338 (Lep.)
——, with —— & Plantilla, 630 (Lep.)
——, —— & Tolentino, J. G., 631 (Lep.)
Wagle, P. M., with Wats & Puduval, 420 (Pl.)
Waisman, H. A., Mickelsen, O., McKibbin, J. M. & Elvehjem, C. A., (797) (Pel.)
Wakil, A. W. & Hilmy, F., 273 (Fev.)
Walker, M. E., with Davis, 640 (R.F.)
Wallace, R. B., 287 *bis* (Mal.)
Wang, C. W., 773 (Leish.)
—— & Chung, H.-L., 769 (Leish.)
——, with Chung, 769 (Leish.)
——, with ——, Liu & Chu, 789 (Mal.)
Warner, D., with Hoyt, 615 (Rab.)
Waterman, J. A., 519 (Vms.)
Watkins, A. M., 518 (Vms.)
Wats, R. C. & Puduval, T. K., 827 (Pl.)
——, Wagle, P. M. & Puduval, T. K., 420 (Pl.)
Watson, R. B. (53) (Mal.)
——, Faust, E. C. & Simmons, J. S., (53) (Mal.)
Watt, A. S., with Richards & Tansley, 699 (Tryp.)
Wax, J. H., with Amolsch, 532 (Misc.)
Weaver, J. W., with Sydenstricker, Geeslin & Templeton, 439 (Pel.)
——, with ——, Schmidt & Geeslin, 437 (Pel.)
Webb, J. L. & Hervel, J. E., 739 (Mal.)
Weber, N. A., 520 (Vms.)
Webster, L. T., 194, 619 *bis* (Rab.)
——, with Casals-Arietm, 622 (Rab.)
Webster, W. J., 577 (Fev.)
Wechsler, Z., 674 (Misc.)
Wegelius, R., 312 (Bb.)
Weinman, D., with Tyzzer, 272 (Fev.)
Welch, N. A. & Jakmauh, P. J., 575 (Fev.)
Wenrich, D. H., 532 (Misc.)
Wense, T., 514 (Vms.)
Wenyon, C. M., 174 (Mal.)
Westphal, A., 372, 870 (Dys.), 677 (Misc.)
Weyer, F., 738, 786 (Mal.)
Whitcomb, E. W., 342 (Lep.)

White, P. B., 719, 720 (Chl.)
White, R. S. & Adhikari, A. K., 741 (Mal.)
——, Aubertin, D. & Smart, J., 468 (B.R.)
Whitfield, F. G. S., 554 (Y.F.)
Whittingham, H. E., 174 (Mal.)
Wickramasuriya, G. A. W., 668 (Mal.)
Wigand, R. (211) (Hel.)
Wigglesworth, V. B., 379 (Misc.)
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